MUSCLE FUNCTION IN RESPONSE TO ROTATIONAL PERTURBATION OF THE GLENOHUMERAL JOINT

By

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DEDICATION

This dissertation is dedicated to the memory of Albert Routledge, Doris Routledge, Russ Hatzel, and Mary Hatzel. Their endless encouragement in the pursuit of my dreams was limitless and their support and belief in me have been unfaltering, and still are, my greatest inspiration for life.

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The glenohumeral (GH) joint has been described as the most mobile and potentially unstable joint in the body. The purpose of this study was to describe the timing (latencies) of muscular firing in normal human subjects versus those with shoulder instability. Subjects making up the control group (N = 5; ht = 70.0 ± 1.41 in; age = 21.6 ± 2.07 yr.; wt = 189.0 ± 27.7 lbs.) were matched to the participating instability subjects by height, weight, age and dominant shoulder. Those that made up the instability group (N = 5; ht = 70.0 ± 1.41 in.; age = 22.0 ± 3.08 yr.; 192.0 ± 29.49 lbs.) presented with a history of subluxation or dislocation to their dominant shoulder. Utilizing fine wire and surface electromyographic (EMG) techniques, subject reflex latencies were measured in response to an external (ER) and internal (IR) rotational perturbation. As the perturbation was applied to the GH joint the subjects were asked to resist the movement and keep the shoulder from moving in the direction of the perturbation. After data collection, one 3 way mixed model analysis of variance (ANOVA) was performed. The between subjects

factor was group (unstable, control), the within subjects factors were muscles investigated (subscapularis, teres major, supraspinatus, and infraspinatus) and movement (IR, ER). An a priori α level of significance was set at $P < 0.05$ for all comparisons. The ANOVA revealed significant differences in the movement, muscle and group main effects, movement by group interaction, muscle by group interaction, movement by muscle interaction and the movement by muscle by group interaction. Significant differences were followed by Tukey HSD post-hoc analysis. The results of this study indicate that the unstable group possessed significantly longer reflex latencies than were noted in the control group for both motions. Given the results of this study, it is safe to assume that significant decrements in stability exist between those with shoulder instability and those without. In conclusion, practitioners must identify these areas of deficiency in the pathological shoulder in order to thwart the negative involvement of the shoulder instability paradigm.

CHAPTER 1
INTRODUCTION

The glenohumeral (GH) joint has been described as the most mobile and potentially unstable joint in the body.[1] Shoulder injuries are frequent and often extensive in sports or occupations during which the arm is moved at a high velocity, under load, or stressed at end ranges of motion. Due to the extensive amount of motion allowed at the GH joint, stability is often compromised for mobility. Although the GH joint allows significant physiological motion, only a few millimeters of humeral head translation occurs during these movements in the normal individual.[2-4] Demanding the utmost in mobility while accommodating relentless force is an essential and dangerous competition between structure and function. Function of the shoulder is dependent on many factors. It is important for clinicians to continue their quest to discover the intricacies of shoulder function and dysfunction.

The GH joint consists of a humeral head articulating with the glenoid one-fourth its size. This feature provides for a unique range of motion and shock-absorbing capability.[5] Stabilizing such an articulation is one of the unique challenges of musculoskeletal anatomy and mechanics. In order for a joint to maintain stabilization during such demanding function, it is imperative to appreciate the involvement of related structures that must work together to contribute to a functional outcome. The interdependence of the GH joint and surrounding structures required to maintain shoulder stability forces the need for researchers to understand all of the anatomical structures in

the shoulder region. Functional shoulder stability is maintained by the integration of static and dynamic mechanisms.

The static mechanism encompasses capsuloligamentous structures such as the joint capsule, labrum, and GH ligaments. These structures function as checkreins to excessive translation or rotation of the humeral head on the glenoid but are lax in the midranges of motion.[6] The GH capsule and ligaments also possess neurosensory characteristics. Mechanoreceptors, imbedded within capsuloligamentous tissue, detect joint loads and transmit sensory information regarding joint motion and position to the central nervous system (CNS).[7-9]

Although four types of receptors are dispersed throughout ligamentous and capsular tissues, Ruffini receptors are the most frequently described.[10] They are considered to behave as both static and dynamic receptors based on their low-threshold, slow-adapting characteristics.[11] In contrast, the low-threshold, rapidly adapting characteristics of Pacinian corpuscles cause them to be exclusively classified as dynamic receptors.[11] Also present in these tissues is Golgi tendon organ-like endings and free nerve endings.[11-14]

Tenomuscular structures contain two types of mechanoreceptors that have received attention for their contribution to joint motion.[15] Golgi tendon organs (GTOs) provide information on muscle tension while muscle spindles encode signals concerning muscle length. Recent theories suggest that the sensory articular receptors and GTOs influence muscle spindle sensitivity and therefore mediate muscle activity and muscle tone.[15]

The sensory information transmitted from mechanoreceptors is utilized by the CNS for conscious and unconscious appreciation of joint motion and position. A complex network of synapses in the spinal cord, brain stem, and cerebral cortex links afferent (sensory) pathways with efferent (motor) pathways. Peripheral receptors not only provide information regarding proprioception and kinesthesia, but also contribute to the involuntary and voluntary muscle activation required for neuromuscular control. The involuntary activation influences muscle tone and muscle reflexes. Voluntary muscle activation results in preprogramming of motor patterns for planned functional activities of the extremity.[9, 15-17] The complexity of the shoulder is necessary to provide efficient, coordinated movement of a joint that is multi-planar and multi-tasked. In order to achieve functional motion of the shoulder, communication, synergism and antagonism amongst all involved structures must be strict. For example, in throwing a ball, particular muscle activation sequences occur in the rotator cuff muscles to ensure that the optimal glenohumeral alignment and compression required for joint stability are provided. These muscle activations take place unconsciously and synonymously with the voluntary muscle activations directly associated with the particulars of the task (i.e., aiming, speed, distance).[18] Proprioceptive information concerning the status of the joint and associated structures is essential for neuromuscular control.

A delicate balance exists between the static and dynamic restraint mechanisms in the GH joint and alterations may evoke shoulder dysfunction. Injury to the static restraints may result in increased laxity and potentially decreased stabilization; meanwhile, injury to the dynamic mechanism may lead to impairments in neuromotor control over the muscles crossing the GH joint. The question exists: what occurs first,

decreases in the integrity of the static restraints leading to a decrement in neuromotor control, or is there a decrease in neuromotor control that causes increased translation, stretching the static restraints and leading to a diminished response of the involved proprioceptors? Identifying the nature of this injury paradigm has been the basis of discussion for many years.

By virtue of the motion allowed at the GH joint and the notion that static restraints provide little support unless tensed, the capsuloligamentous structures are unable to prevent excessive GH translation. It has been demonstrated that mechanoreceptors are more active at the end ranges of motion;[19] if the end range is extended due to the envelope of motion or they simply are not stressed until later in the range, then problems in the function of the notification of the dynamic structures to fire for stabilization may occur. When these mechanoreceptors fire as they become tensed, a force is exerted on the humeral head to assist in stabilization. Capsular distention may be the result of injury in response to dislocation, subluxation or micro-traumatic throwing episodes. Smith and Brunolli[20] have observed deficits in shoulder kinesthesia and joint position sense in male subjects with unilateral, traumatic, recurrent anterior shoulder instabilities. Decrements in proprioceptive communication due to structural damage may precipitate instability pathologies and result in decreased stabilization during functional activity. Examination of pitchers with anterior glenohumeral instability by Glousman et al.[21] revealed significant pattern changes in all muscles tested about the shoulder except the deltoid. A notable change identified by Glousman et al.[21] was that in those with instability a decrease in internal rotator activity was seen. This may result in further external rotation of the humerus in the power phases of the pitch, thus

perpetuating and/or producing further humeral translation and instability. In the complex shoulder, distention of the joint capsule or injury to dynamic stabilizers may result in a delayed reaction to preparatory and reactive muscle activation.[22] These conditions may lead to increased humeral translation during functional tasks, ultimately leading to repetitive injury. This phenomenon represents inadequacies in the neuromuscular function of the shoulder, possibly leading to the inauguration of debilitating shoulder problems.

At this point it is important to distinguish between laxity and instability, as many clinicians confuse these very different phenomena. Shoulder laxity is described as the ability to passively translate the humeral head upon the glenoid fossa. Meanwhile, shoulder instability is a clinical condition in which unwanted humeral head translation compromises the comfort and function of the shoulder.[23] It is common to identify lax shoulders that are completely asymptomatic, in high performance athletes. For instance two patients may have equally "lax" shoulders, yet one will have symptomatic instability while the other is unaffected. This points to the importance of dynamic muscle function to compensate for deficits within the static restraint mechanism.

The involuntary and reactive characteristics of muscles are responsible for maintaining joint integrity after expected or unexpected joint perturbation. A perturbation is an event or condition that creates a disturbance or agitation to the intended task, and is unexpected. The recruitment of muscle fibers and utilization of stored elastic energy is critical for balancing intrinsic and extrinsic joint loads.[24] If muscles are to provide dynamic restraint, stored elastic energy and motor unit recruitment must combine to develop a sufficient magnitude of force in a timely manner, resisting excessive loads.[24]

Reflex pathways from articular receptors can elicit involuntary muscle activity under high loads, but also have a strong influence on the sensitivity of muscle spindles. Muscle spindles then have an excitatory effect on agonist muscles via stretch reflexes, while GTOs inhibit agonist muscle activity due to excessive muscle tension.[25] Therefore, the reactive capabilities of muscle for providing dynamic restraint are dependent on equilibrium between excitatory and inhibitory influences from muscle spindles and GTOs.[25]

GH joint movement patterns are planned in the cerebral cortex based on past experiences and muscles are recruited in preparation for joint loads. Preprogrammed muscle activity is responsible for facilitating the tenomuscular unit in anticipation of joint movements or loads and is necessary for high speed, ballistic type movements.[26] Once a motor program has been developed in the cortex, dynamic stabilization occurs faster resisting excessive joint motion and assisting with dynamic function. It is essential to have timely dynamic responses in order to counteract the extremes of the range of motion during functional tasks.

The GH joint is minimally constrained and relies significantly on neurologic influence and motor control for dynamic stabilization and would seem to be the most productive site to study dynamic stability.[27] As indicated earlier, muscular activity is significantly altered in those shoulders that are unstable.[21] The issue may not necessarily be the amount of force differences between those who are unstable and those who are normals, but in all likelihood it is the timing of the reflexes that may be responsible for excessive joint motion during throwing. Latimer et al.[28] investigated the reflex response times in response to an anterior translation force. The data indicated that the anteriorly

located muscles (pectoralis major, anterior deltoid, upper subscapularis, and long head of the biceps) all responded in 110 to 160 msec. They also indicated that anteriorly located muscles preceded the posteriorly located muscles. The timing of latencies is essential in maintaining dynamic stabilization. Inhibition of internal rotation and protraction of the scapula accentuate the stresses on the anterior restraints, thereby leading to increases in laxity and causing the continuum of the instability vs. laxity paradigm. The reflex arc of the shoulder in response to an anterior translation force has not been studied in those with shoulder instability. The length of this neural pathway is relatively shorter than other joint reflex neural pathways due to its shorter distance, and therefore muscle activation may be able to stabilize the GH joint before subluxation or dislocation occurs.

Statement of the Problem

Stabilization of the GH joint is essential in the maintenance of fluid and efficient motion at the shoulder. Efficient communication of the involved restraint mechanisms is requisite in the maintenance of stabilization within the GH joint. During functional activities such as throwing, timely adjustments in muscle function are required to provide fluid and efficient function, meanwhile avoiding injury. The problem exists in identifying the specific mechanism that precipitates the miscommunication of the restraint mechanisms thereby leading to instability and related shoulder pathology. By identifying the specific cause for repeated bouts with the unstable shoulder, clinicians and rehabilitation professionals could potentially combat related pathologies. The identification and development of exercise protocols aimed at thwarting the cause of instability could help to avoid injury of the GH joint. Through an extensive literature

review, no studies were found that identified differences in reflex stabilization between those with shoulder instability as compared to the normal shoulder.

The purpose of this study is to describe the timing (latencies) of muscular firing in normal human subjects versus those with shoulder instabilities of the GH joint when an internal and external rotation perturbation is applied.

Research Hypotheses

The following hypotheses will be tested in this study:

1) Significantly longer latencies in response to the ER perturbation will be apparent in the instability group as compared to the subjects without shoulder instability (control group).

2) Significantly longer latencies in response to the IR perturbation will be apparent in the instability group as compared to the subjects without shoulder instability (control group).

Definition of Terms

The following terms are used in the dissertation.

Amortization phase. For the purposes of this investigation, the amortization phase represents the time between the eccentric and concentric phase of muscle action. When a muscle is stretched prior to movement, the force that is stretching the limb must be overcome before the muscle can produce a movement. The amortization phase exists at the end of the eccentric muscle action and continues until the beginning of the concentric muscle action. It is also referred to as the latency phase.

Concentric muscle action. In a concentric contraction the muscle shortens in length while tension develops to overcome resistance.[29]

Eccentric muscle action. During an eccentric action, the resistance is greater than the muscular force being produced, and the muscle lengthens while producing tension.[29]

External rotation (ER) at 90/90. External rotation at 90/90 occurs when the subject reaches maximum external rotation with the humerus positioned at 90° of GH abduction.

Electromyography (EMG). Electromyography is defined as the preparation, study of, and interpretation of a graphic record of the contraction of a muscle as a result of electrical stimulation.[30]

Fine wire electrodes. Fine wire electrodes are described as the use of wire electrodes (50 micron) with 2 to 3 mm bared tips that are inserted into the specific muscle to be studied. These electrodes allow for in vivo study of muscle activity in response to the perturbation. Fine wire electrodes minimize interference and allow for a more enhanced examination of muscular activity.

Glenohumeral joint (GH joint). The GH joint pertains to the articulation of the humerus and the glenoid cavity of the scapula.[30]

Internal rotation at 90/90. Internal rotation at 90/90 occurs when the subject reaches maximum internal rotation with the humerus positioned at 90° of GH abduction.

Latency. For the purpose of this project, latency represents the "turn around time" that a muscle undergoes in order to resist a force and create a positive outcome. It is also related to amortization time.

Perturbation. A perturbation represents a state of being disturbed or agitated.[30] In this case it represents the act of delivering an unexpected force to the GH joint. This force is provided from a specially developed perturbation device and results in the GH joint moving into ER and IR.

Reflex stabilization. The term reflex stabilization refers to the muscular activity (voluntary, involuntary) that is evident after a perturbation. Reflex stabilization is responsible for helping to maintain the integrity of the GH joint.

Shoulder instability. For the purposes of this investigation, those designated as having shoulder instability are those subjects who present with a history of either a dislocation or subluxation of their dominant GH joint.

Surface electrodes. Surface electrodes are placed on the skin with an adhesive substance. The electrodes are 1.0 cm in diameter silver/silver chloride electrodes embedded in an epoxy-mounted preamplifier. These electrodes allow examination of muscular activity but are placed on the exterior of the skin.

90/90 position. Refers to positioning of the GH joint with the humerus at 90° of humeral abduction and 90° of humeral external rotation.

Assumptions

1. All subjects were honest and accurate when stating their eligibility for participation.
2. The groups (control, instability) were representative of their population.
3. Each subject performed all tests using maximal effort in the shortest amount of time as possible.
4. The ER and IR pertubation trials represent that which exists during functional activity while throwing a ball.
5. Placement of the fine wire electrodes did not affect muscular function.
6. Measurements of muscle latencies are an accurate assessment of reflex stabilization.

Limitations

1. Only one velocity was used for the perturbation application; therefore, the ability to predict subject performance at velocities other than those in this study may be limited.

2. The population for the instability group is structured on the specific definition for instability as it relates to this study. It includes anyone who has ever dislocated or someone who experiences or has experienced a subluxation episode of their dominant GH joint.

3. Those that make up the control group are those that have not experienced a dislocation or subluxation episode. They may have history of other shoulder pathologies although they must be asymptomatic at the time of testing.

4. The perturbation device is only a representation of what occurs during functional activity (throwing motion).

5. The perturbation in the ER and IR directions will be applied with the subject positioned at 90% of their maximal external rotation.

Significance

This study may help determine the parameter characteristics inherent to a population with shoulder instability. Through a thorough literature review, there have not been any studies that have compared muscle latencies in response to an ER and IR perturbation between those with and without shoulder instability. This study contributes to science by identifying a variable (latency), which may add to the understanding of shoulder instability and enhance the criteria. An athlete with deficiencies in muscle latencies resulting in altered muscle firing patterns may have an increased chance for shoulder pathology. The results of this study have clinical implications in regards to rehabilitation and the prevention of injury.

CHAPTER 2
REVIEW OF LITERATURE

The evaluation of shoulder dysfunction is a challenging task requiring an in-depth knowledge of the anatomy, biomechanics, and stabilization strategies of normal and abnormal shoulder motions. The GH joint is inherently unstable and exhibits the greatest amount of motion found in any joint in the human body.[31] Matsen et al.[5] demonstrated excessive passive displacement (10mm inferiorly and 8mm anteriorly) in normal asymptomatic shoulders. Because of the excessive motion that occurs at the glenohumeral joint, both ligamentous and surrounding musculature are simultaneously responsible for stabilization. The purpose of this section is to examine the stabilizing structures and underlying mechanisms of maintaining GH joint stability.

Static Stabilizers

Bony Geometry

The bony geometry of the GH joint can be an important source of GH joint stabilization. This articulation is made up of the humerus and the glenoid of the scapula. The articulating surface of the glenoid is much smaller than that of the humeral head, and the surface area of the humeral head is approximately three to four times that of the glenoid.[32] The surface area ratio between the humeral head and glenoid has been referred to as the GH index (maximum diameter of the glenoid/maximum diameter of the humeral head).[33, 34] This ratio is approximately 0.75 in the sagittal plane and approximately 0.6 in the transverse plane.[33] Additionally, at any given time during normal motion, only 25-

30% of the humeral head is actually in contact with the glenoid.[35-37] This lack of articular contact contributes to the inherent instability of the GH joint.[38] The GH index is a number that is calculated by using only bony measures specific for this joint. It is important to note the presence of cartilage within the glenoid fossa.[39] Matsen et al.[39] noted that the articular cartilage of the glenoid fossa is thickest in the periphery and thinnest in the center. Thus, the glenoid joint surface may be more concave than the underlying bone would first indicate, and, thus the GH joint may be slightly more congruent than it appears on radiographs.[6] The cartilage within the glenoid fossa is identified as the glenoid labrum.

Glenoid Labrum

The glenoid labrum is a fibrous structure that forms a ring around the periphery of the glenoid and acts as an anchor point for the capsuloligamentous structures. In addition, it contributes to functional stability by increasing the depth of the glenoid fossa. Howell and Galinat[40] proposed that the stabilizing effect of the labrum was analogous to that of a "chock block," preventing the wheel of a tractor from rolling down a hill. It has also been proposed by Soslowsky et al.[32, 41] and by Bowen et al.[42] that the labrum may also contribute to stability by increasing surface area and acting as a load bearing structure for the humeral head. Several investigators have also reported that a small amount of fibrocartilage exists at the junction of the glenoid and fibrous capsule.[43-45]

Cooper et al.[46] demonstrated significant anatomic variability in the labrum around the periphery of the glenoid. In its superior portion above the equator of the glenoid, the labrum quite frequently is attached loosely and may function more as a mobile extension

of the glenoid surface than as a rigid "chock block." Below the equator of the glenoid the labrum consistently attaches to articular cartilage.

During arm movements, the humeral head articulates with the periphery of the glenoid and may actually articulate with the capsulolabral structures as well.[32] This represents an integration of labrum and capsuloligamentous structures responsible for static stabilization of the GH joint. Bankart,[47, 48] expounding on a concept originally published by Perthes,[49] reported the "essential lesion" responsible for shoulder instability was the detached labrum and capsule from the glenoid (referred to by subsequent authors as a Bankart lesion).[6]

Capsuloligamentous Structures

The shoulder joint capsule is comprised of multiple layers of collagen bundles. Collagen bundle orientation is comprised of radial fibers that are linked to each other by circular elements.[6] Radial fibers provide a direct connection between the humeral head and the glenoid while the circular collagen elements serve as a connection between radial fibers. The circular orientation in fact plays a significant role in providing joint stability. Thus, rotational forces produce tension within the fibers, which leads to compression of the joint surfaces but also a centering of the joint.[50] Compression is achieved by the unique capsular make-up, which results in fiber cross linking during rotational movements. Distractive force leads to an increase in longitudinal stretching of the capsule and, thus, constriction of the cylinder, resulting in greater joint compression and enhanced effects of negative intraarticular pressure.[50]

The capsule is also reinforced by capsular ligaments, which help to greatly assist in joint stability. Historically, the GH ligaments have been described as thickenings in

the capsule of the shoulder joint.[51-55] The anterior GH joint capsule is composed of three distinct ligaments: the superior glenohumeral ligament (SGHL), the middle glenohumeral ligament (MGHL), and the inferior glenohumeral ligament (IGHL) complex.[56-58] The SGHL arises from the anterosuperior labrum anterior to the biceps tendon and inserts superior to the lesser tuberosity. The MGHL originates adjacent to the SGHL and extends laterally to attach on the lesser tuberosity with the subscapularis tendon. The IGHL complex is composed of three functional portions: an anterior band, a posterior band, and an axillary pouch. Each of these structures exhibits a large amount of variability, with the MGHL exhibiting the greatest degree of variation.[56, 59] Posteriorly, the capsule is thinnest and has no capsular ligaments except for the posterior band of the IGHL complex.

The role the shoulder capsule and anterior GH ligaments play in stabilizing the GH joint is complex and varies with both shoulder position and the direction of the translating force.[6] The constraints to inferior translation of the humeral head on the glenoid vary based on arm position and degree of rotation. Blasier et al.[60] and Terry et al.[61] classify the structures in the direction of the translation as the primary restraints and the structures on the opposite side of the joint as the secondary restraints. Warner et al.[4] have demonstrated that the primary and secondary restraints preventing inferior translation with the arm in the adducted position are the SGHL and the coracohumeral ligament. When the arm is abducted to 45° and neutrally rotated, the anterior band of the IGHL complex is the primary restraint to inferior translation. At 90° of abduction, the posterior band of the IGHL complex is the primary stabilizer against inferior motion of the humeral head on the glenoid.

The anterior GH ligaments function as primary restraints to anterior translation.[62,63] The superior and middle glenohumeral ligaments are the primary restraints to anterior translation with the arm fully adducted.[64] The MGHL plays a significant role in limiting anterior translation within the midrange of shoulder abduction.[59, 64, 65] The IGHL complex, particularly the anterior band, is responsible for preventing anterior translation of the humeral head with the arm abducted to 90° or greater.[62, 63]

The concepts to posterior translation are also based on arm position.[6] O'Brien et al.[62] have reported that the IGHL complex (with the posteroinferior capsule) is the primary passive stabilizer against posterior instability with the arm in 90° of abduction. In contrast, when the arm is positioned below 90° of abduction, the posterior capsule provides the primary restraint to any posterior force.

As a general rule, the superior capsular structures play a significant role in joint stability when the arm is adducted.[6] Conversely, the inferior structures are preeminent in providing joint stability from 90° of abduction toward full extension.[6] O'Connell et al.[19] recorded the amount of strain present within the GH ligaments in various arm positions. They reported a concentration of strain in the IGHL complex at 90° of GH abduction; at 45°, the IGHL complex and MGHL developed the highest strain; and at 0° of abduction, strain was concentrated within the superior and middle glenohumeral ligaments.

Rotator Interval

The "rotator interval" comprises the region between the anterior border of the supraspinatus tendon and the superior border of the subscapularis tendon.[66] The SGHL and coracohumeral ligament are located in this region of the capsule. There is a large

anatomic variation in the size of the rotator interval found in individuals, ranging from a narrow interval to large lesions, where most or the entire rotator interval is absent.[67] Nobuhara and Ikeda[68] have reported an association between inferior instability and a large interval. Harryman et al.[69] reported this portion of the capsule plays a significant role in preventing inferior subluxation in the adducted shoulder and is a secondary restraint to posterior translation.

Intraarticular Pressure

The GH joint surrounded by the capsular structures is airtight, helping to enhance stability. Kumar and Balasubramaniam[68] were the first to observe that venting the joint capsule of a cadaveric shoulder resulted in marked inferior translation of the adducted arm. The normal intraarticular pressure is negative, creating a relative vacuum that resists GH joint translation.[70-72] Because the rotator cuff is not contracting when the arm is hanging at the side, negative intra-articular pressure constitutes a major mechanism preventing inferior subluxation in this position. Browne et al.[70] demonstrated the average intra-articular pressure in an adducted cadaver shoulder to be -42 cm H_2O, and the application of a 25-N inferior force caused the pressure to drop to -82 cm H_2O. If these properties are disrupted by puncture or tear in the capsule, subluxation may occur. Wuelker[73] reported venting the capsule increased displacement by 47% anteriorly, 49% posteriorly, and 61% inferiorly. In addition, Habermeyer et al.[74] noted that the vacuum effect of viscous and intermolecular forces was lost in the unstable shoulder (Bankart lesion) and that the labrum acted much like a seal or gasket. In effect, once a labral injury occurs, it results in atmospheric pressure changes and a loss in passive GH joint stability. A return to normal intra-articular pressure after injury has not been reported in

the literature, and it is believed that once lost it does not return. It should be noted that although the intra-articular pressure of the GH joint does offer static support, muscle contraction and tension in the ligaments during active motion of the joint have a much greater role in stability.

Dynamic Stabilizers

The dynamic stabilizers of the GH joint play an active role in maintaining stability during activity. The primary active stabilizers include those of the rotator cuff (supraspinatus, infraspinatus, teres minor, and subscapularis), long head of the biceps, and the deltoid. The function of these dynamic stabilizers is to compress the articular surface of the humerus in the glenoid. Stabilization is provided by the following mechanisms: (a) joint compression, (b) coordinated contraction to center the humeral head into the glenoid, and (c) dynamization of GH ligaments through cuff attachments.

These muscles act together to provide movement of the arm but they also function to stabilize the GH joint. By utilizing an agonist/antagonist relationship, for example as the subscapularis fires in the late cocking phase of throwing, the infraspinatus and teres minor fire to create a vector opposite that of the subscapularis. This agonist/antagonist relationship is referred to a force couple. Inman[23] first described the force couple relationship within the glenohumeral joint. A force couple, by definition, occurs when two parallel forces of equal magnitude but opposite in direction are applied to a structure at equal distances from the center of mass.[23] The two force couples noted at the shoulder are the subscapularis counterbalanced by the infraspinatus/ teres minor, and the deltoid counterbalanced by the inferior rotator cuff muscles.[23] (Figure 2.1) Experimental studies

by Bowen et al.[75] and Lippitt et al.[76] have shown that the magnitude of this stabilizing effect greatly exceeds that of the static capsuloligamentous factors.

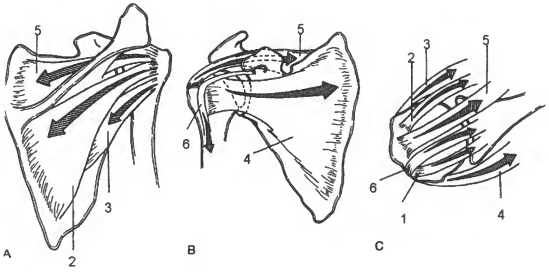

Figure 2.1. Dynamic joint stability. Description of the Inman force couple. A) Posterior view, B) anterior view, and C) superior view. Anterior deltoid (1), infraspinatus (2), teres minor (3), subscapularis (4), supraspinatus (5), and long head of the biceps (6).[6]

Lippitt et al.[76] reported joint compression resisted up to 60% of the transglenoid force. In addition, Bowen et al.[75] have noted that a joint compression load of 111 N continued to stabilize the shoulder despite a 50-N displacement force and sectioning of three-fourths of the joint capsule. Increasing the compressive force on the humeral head results in a more "centered" position on the glenoid, reducing the magnitude of translations. When these forces are not balanced or equalized, between either the prime movers or appropriate force couple, abnormal GH mechanics occur. In the throwing athlete, clinically they present with a decrease in strength in the posterior musculature, resulting in a loss of stability secondary to a force couple imbalance between the anterior and posterior cuff musculature.[77, 78]

Cain et al.[79], in cadaveric experiments, demonstrated that maximal contraction of the posterior rotator cuff muscles reduced anterior ligamentous strain. Blasier et al.[60] biomechanically studied the role of the rotator cuff and concluded that the muscles of the entire cuff contributed to stability equally. If tension on any one to the entire rotator cuff was omitted or significantly reduced, there was a substantial reduction in anterior joint stability compared with when tension was applied to all components.[6] The dynamic stability of the GH joint is accomplished through the combined efforts of all the muscles around the shoulder stabilizing the humeral head within the glenoid via compressive forces.

Neurological Components

During functional activity it is necessary to keep the humeral head within the glenoid. This is accomplished by maintaining communication between the dynamic stabilizers attempting to steer the humeral head while it maintains its integrity within the glenoid. The principle of this dynamic communication is referred to as "neuromuscular control".[80, 81] This process is highly dependent upon the continuous interplay of afferent and efferent information. Sensory information (proprioception) travels through afferent pathways to the CNS, where it is integrated with input from other levels of the nervous system, eliciting efferent motor responses (neuromuscular control) vital to coordinated movement patterns and functional stability.[82] Myers and Lephart[82] suggests that a contemporary interpretation of proprioception be defined as the afferent information concerning the three sub-modalities of joint position sense, kinesthesia, and sensation of resistance. Each of these sub-modalities can be appreciated both consciously and unconsciously in mediating neuromuscular control.

Proprioceptive information originates at the level of the mechanoreceptor or "proprioceptor."[83] Mechanoreceptors are sensory neurons or peripheral afferents present within the muscle, tendon, fascia, joint capsule, ligament, and skin about a joint.[84, 85] Deformation to the tissues in which the mechanoreceptors lie causes a mechanically gated release of stored sodium, eliciting an action potential.[86] As an increase in tissue deformation is noted an increase in action potential is noted, thereby increasing input to the CNS.[84, 86]

Recently, Vangsness et al.[85] reported that ruffinian, pacinian corpuscles and golgi mechanoreceptors are located within the capsuloligamentous structures in the shoulder. Ruffini afferents are believed to be stimulated only with extremes of motion through tensile force, acting as limit detectors.[84] Like ruffini receptors, pacinian corpuscles respond to extremes of motion but through both compressive and tensile mechanisms rather than stretching alone.[84] Because the capsuloligamentous structures of the shoulder are reported lax in mid ranges of motion[87, 88], mechanoreceptors present within the joint capsule and ligaments are believed to contribute proprioception information when maximal deformation occurs at end ranges of motion.[84, 89] In addition to the capsuloligamentous mechanoreceptors, the musculotendinous mechanoreceptors (Golgi tendon organs) play an equally important role in proprioception. At the tendinous region of the muscle, the tension-sensitive GTOs are recruited when muscle contraction pulls on the tendon, relaying afferent feedback concerning joint position and musculotendinous tension.[90, 91] As a protective mechanism, stimulation of the GTO facilitates relaxation of the agonist muscle under tension while eliciting contraction of the antagonist muscle group.[91]

An important aspect of dynamic stabilization and perhaps increasingly important in the throwing athlete is reflex stabilization. Several investigators have demonstrated that a reflex exists between fibrous joint capsule and musculature in the feline GH joint.[92, 93] In this instance, the stabilizing structures are deformed upon application of resistance to the joint eliciting a feedback and concluding with muscle contraction. The problem exists in the time needed to elicit this muscle contraction. If the reflexive muscle contraction does not respond in time to prevent the humerus from translating outside the glenoid, mechanical failure (i.e., dislocation, subluxation) may be encountered.

The phases of late cocking and early acceleration, during which the humerus is in abduction and maximum external rotation, are the critical stages at which greatest stress is placed on the anterior restraints of the shoulder. An increase in latency phase during the throwing motion will lead to miscommunication and inadequate stabilization by the dynamic mechanism. The time that makes up the interval between eccentric contraction and concentric contraction makes up the latency phase, this phase is also known as the amortization phase. This neuromuscular imbalance may occur as part of the etiology of the syndrome of anterior instability of the shoulder.[21] If the imbalance occurs secondary to instability, it increases and propagates the syndrome.

The blending of the rotator cuff tendons into the shoulder capsule provides assistance to the required communication between the musculotendinous (dynamic stabilizer) and the capsuloligamentous (static stabilizer) structures. Warner et al.[4, 94] and others[19, 95-98] have shown that, at the extremes of rotation, these static structures function in a load sharing manner to prevent excessive translation and rotation. Clark et al.[99] have

demonstrated that the rotator cuff tendons are attached to the humerus directly through portions of the capsule and ligaments and have proposed the possibility that rotator cuff contraction causes tension in the ligaments, thereby "dynamizing" these structures during active GH rotation. As the rotator cuff musculature contracts, tension is produced within the capsular ligaments, actively tightening the GH ligamentous capsule, again promoting centering of the humeral head within the glenoid fossa.[6]

Neuromuscular Control

In order to maintain appropriate stabilization of such an inherently unstable joint, it is prerequisite to possess efficient communication between responsible stabilization mechanisms. It is the intention of this section to present an explanation of the events that occur to assist in the adequate stabilization of the glenohumeral joint. Below you will find descriptions of the multiple pathways and interactions that simultaneously contribute to functional stability.

Motor function whether being for locomotion or attempting to elicit a fine motor task, requires a complex system of communication. This communication is commonly referred to as proprioception. Unfortunately, this is a misnomer; proprioception as described by Sherrington[83] is a form of communication involving afferent pathways. Only sensory information received from proprioceptors within the affect area of movement provides information regarding postural equilibrium, joint stability and muscle sense.[100] The receptors for proprioception are located in the skin, joints, ligaments, muscles, and tendons and are activated by changes in pressure and movement of the soft tissue structures. While proprioception is essential for movement and the completion of functional tasks, it should be stressed that the proprioceptive information is solely

afferent in nature. The input provided from the proprioceptive system is transported to the CNS in order to generate a motor response. Control of movement and posture is dependent on a continuous flow of sensory information about events in the environment.[101] Motor responses facilitated by the proprioceptive system originate at three levels of motor control: (a) the spinal cord, (b) the lower regions of the brain, and (c) the cerebral cortex.[82, 102] Activity at the spinal level is the final route for all movements. These movements include voluntary and reflex motor actions. Before investigating the three levels of motor control it is essential to understand the path of transmission of afferent and efferent information. Motor control at the spinal level is the result of a variety of influences from the descending pathways from the brain, and from the proprioceptors in the muscles and tendons themselves.

Spinal Cord

Lower Motor Neurons

The lower motor neurons form the final route to the muscles in all movement, both voluntary and reflex. The lower motor neurons exist within the anterior horn of the spinal cord, while the axons from these neurons lie in the peripheral nerves supplying the muscles.[103] Lower motor neurons are primarily stimulated by activity in the descending pathways, which terminate in the anterior horn of the spinal cord at all levels.[103] These motor neurons are also stimulated by the activation of local spinal reflexes.

Before an efferent signal can be transmitted to the motor unit to produce an action, the information must be transmitted along and throughout the spinal cord. The spinal cord is composed of tracts of nerve fibers that allow a two-way network of nerve impulses. One direction of the network is provided by the afferent fibers which carry

different signals from the mechanoreceptors to the upper levels of the CNS.[104] The other pathway is made up of efferent pathways initiated from the cerebrum and descend down to the muscles for activation. The spinal cord acts as a facilitating pathway between both sensory and motor fibers between the brain and the periphery.[104]

Within the spinal cord exist two types of neurons responsible for communicating information to the brain and periphery. Located in the anterior horn of the cord gray matter, the anterior motor neurons give rise to the nerve fibers that leave the cord through the anterior roots and innervate the skeletal muscle fibers.[105] These anterior motor neurons consist of two types - alpha motor neurons and gamma motor neurons.

The alpha motor neurons are made up of large type A nerve fibers, which are responsible for innervating large skeletal muscle fibers.[104] The number of muscle fibers stimulated by a single motor nerve characterizes a motor unit. The gamma motor neurons are responsible for transmitting impulses through A gamma fibers in the muscle spindles.[104] Gamma motor neurons are constantly influenced by the mechanoreceptors of the joints and the descending motor neurons.[104] The whole gamma motor neuron loop (gamma motor neurons-muscle spindles- primary muscle spindle afferent pathways) may contribute to the preprogramming of the stiffness of muscles around the joint and thereby also to the regulation of joint stiffness and joint stability.[105] Gamma motor neurons consist of two classes, one controlling the dynamic sensitivity of the muscle spindles and the other controlling the static sensitivity.[104]

When the motor units are stimulated after the impulse has traveled to the spinal cord, the extrafusal fibers are stimulated by the large diameter alpha motor neurons.[103] The large diameter alpha motor neurons, which supply the extrafusal fibers, consist of

two types. The large diameter alpha motor neurons supply the fast Type II muscle fibers active in rhythmic motion.[103] The smaller diameter alpha motor neurons supply the slow type I muscle fibers involved in tonic postural activity.[103] This complete pathway is sometimes called the fusimotor loop and the reflex activity is known as the stretch reflex.[103]

Spinal Reflexes

The two primary spinal reflexes include the myotactic and Golgi tendon reflex. The myotatic unit is responsible for maintaining a muscle at constant length to hold a position. The myotatic unit relies on information from receptors that lie parallel to the muscle fibers and are known as muscle spindles.[103] Each of the muscle spindles consists of a capsule of connective tissue, which encloses a grouping of small muscle fibers known as intrafusal fibers.[103] Two types of intrafusal fibers exist, including the nuclear bag fibers and the nuclear chain fibers. The nuclear bag (dynamic) fibers respond to rapid changes in length of muscle, while the nuclear chain (static) fibers respond to prolonged slow stretch.[103] In the middle of the grouping of intrafusal fibers are the nuclei that are non-contractile. Around this central area (nuclei) is the annulospiral ending, which is the primary sensory ending of the muscle spindle.[103] Impulses from the annulospiral ending pass along the sensory neuron into the spinal cord where they stimulate the motor units of the same muscle.[103]

The intrafusal fibers that make up the muscle spindle are themselves contractile and are supplied by small diameter gamma motor neurons originating in the spinal cord.[103] The stimulation of these intrafusal fibers causes them to contract. When this

occurs the spindle shortens, which makes the muscle spindle more sensitive to distortion and reflex activity is increased.[103]

Muscle spindles are slow adapting mechanoreceptors dispersed between the extrafusal muscle fibers and are responsible for detecting changes in muscle fiber length and rate of length change.[84] When stimulated by length changes, muscle spindles are capable of static and/or dynamic responses.[104] Static response occurs when the muscle spindle is slowly stretched, this may be affected by frequency of sensory impulses which directly relates to the magnitude of the stretch. [104] The dynamic response of muscle spindle also occurs when there is a length change, but transmission of the dynamic response will continue only while the muscle is changing length. [104] Excitation of the muscle spindle by gamma motor nerves, does not function as part of the contractile component, but adjusts the length and tension on the receptor portion of the muscle spindle.[100] This mechanism regulates the sensitivity of static and dynamic responses of the muscle spindle to length changes.[91] By modifying the sensitivity of the muscle spindle, sensory information can be transmitted quickly through afferent firing to the central nervous system with information pertaining to the muscle length and rate of change in length.[91] Muscle spindles also have the capability to offer joint stability by mediating involuntary of muscle activity referred to as the stretch reflex mechanism. The spinal level interprets afferent information at the unconscious level, and through reflexes, mediates involuntary activity.[104] It is believed that these reflexes integrate the activation and/or inhibition of muscles, providing joint stabilization and movement.[100] The reflex activity provides fast motor responses to afferent information.

Another method of regulating stability at the spinal level is that of the Golgi Tendon Reflex (GTO reflex). The GTO reflex is a negative feedback inhibitory reflex that prevents muscle tension from becoming too high, helping to avoid injury.[91] As does the myotatic stretch reflex, the GTO reflex includes both a dynamic and static response. The dynamic response occurs when the muscle tension suddenly increases, for example when an unexpected stretch occurs.[91] The static response occurs when the muscle tension settles down to a lower level of steady state firing.[91]

Upper Motor Neurons

The neurons that are responsible for the link between the brain and the spinal cord are known as upper motor neurons.[103] These upper motor neurons reside in the descending tracts of the brain stem and spinal cord. The output that is provided from the upper motor neurons influences the activity in the lower motor neurons and spinal cord, which provide the control for the active muscles during movement.[103] The brain stem serves as a site for the collection of command signals from higher levels, which command the brain stem to modify specific control functions through the body.[91] In addition to controlling specific muscle action, the brain stem also plays an integral part in controlling body movement and equilibrium.[91, 103]

Lower Regions of the Brain

Several descending pathways are directly or indirectly under the control of the brain stem, basal ganglia, and the cerebellum. Each hierarchical level receives information from the periphery so that the sensory input can modify the action of the descending commands. These different areas are also essential for normal motor function.

Brain Stem

Motor control from the brain stem integrates visual and vestibular information and primarily functions in the maintenance of balance and posture.[17] This level of motor control also unconsciously promotes the balance of compressive and shearing forces on the shoulder through involuntary muscle activity.[17] Located within the brain stem are various motor nuclei that serve as a relay point forming polysynaptic routes to the lower motor neurons.[103] The tectum of the midbrain consists of two nuclei that are responsible for visual and auditory information.[103] Also located within the midbrain is the red nucleus which is a motor nucleus that provides a link between the cerebellum and the lower motor neurons since there is no direct descending pathway from the cerebellum to the lower motor neurons.[103] Located within the medulla is the vestibular nucleus which receives input from the vestibule of the ear. The descending tract from the vestibular nucleus links to the lower motor neurones of the extensor muscles which provide support for the body as the head turns.[103] As for the reticular formation, this extends along the core of the brain stem and is a collection of nuclei that are closely related.[103]

Unique from the other nuclei within the brain stem the reticular formation receives ascending somatosensory information from the spinal cord, and also descending fibers from the cerebral cortex.[103] The medial and lateral groups within the reticular formation are thought to be primarily responsible for support and balance of the body during movement.[103] From the brain stem the descending tracts synapse with skeletomotor (alpha) and fusimotor (gamma) neurons at the spinal level.[103] The main function of the rubrospinal and lateral reticulospinal tracts is in positioning and support by the proximal muscles of the limbs during movement.[103] For example, the muscles

around the shoulder region, the elbow and wrist, which support the upper limb during fine manipulation movements of the hand, are mainly activated via these routes.[103] The balance and posture of the body is maintained through the communication within the vestibulospinal and medial reticulospinal tracts, they are mainly activated in order to counteract the effects of gravity on posture.[103]

Basal Ganglia

The basal ganglia are found at the base of the cerebral hemispheres and in the midbrain. The basal ganglia serve as part of the lower brain. The basal ganglia include the caudate, putamen, globus pallidus, subthalamic nucleus and the substantia nigra.[103] Nerve fibers link the individual nuclei with each other to form a complex interdependent system, which functions as a whole. Most of the input to the basal ganglia is from the motor areas of the cerebral cortex, and the basal ganglia project back to these areas of the cortex via the thalamus.[103] The main influence of the basal ganglia on movement is via the motor cortical areas and their output to the motor neurons of the spinal cord in the planning and execution of movement. The basal ganglia have no direct link with the spinal cord. Their influence on the lower motor neurons is via descending pyramidal and extrapyramidal pathways from the cortical motor area.[103]

Cerebellum

The cerebellum is crucial to the control of all rapid and complex muscular activities. It plays a major role in the timing of motor activities and in rapid progression from one movement to the next. The overall function of the cerebellum is the control of the performance of smooth, coordinated movements with correct timing and sequence.[104] As the cerebellum compares the intended program of muscle contraction from the

specific motor areas with continuously updated sensory information from the peripheral parts of the body, it acts as an integration system.[106] Information is communicated the feedback regarding the movements of the body and adjusts movements as deemed necessary to meet the environmental situations.[106] All incoming information about the exact tension and position of the joints, muscles, and tendons and the body's position are noted relative to the environment, and then it determines the correct plan of action to produce the desired movement.[106]

The cerebellum cannot initiate movement, but coordinates and regulates muscle activity. The cerebellum decides, based on the input from the periphery, what is the best plan of action to accomplish this movement. Additionally, the cerebellum aids the cerebral cortex in planning the next step of movement in advance while the current movement is still performed, thus helping to progress smoothly from one movement to the next.[104] The cerebellum is able to identify inadequacies of the intended movement and adjust future tasks accordingly. The specific movements are made stronger or weaker after an error, adapting the excitability of the appropriate cerebellar neurons.[104] Future muscle patterns will be better mediated with this ability to retain previous movement information.

Cerebral Cortex

The cerebral cortex is located at the outer portion of the cerebral hemispheres and is responsible for registering sensory stimuli allowing for voluntary control of specific movements.[91] The cerebral cortex controls complex movement patterns and is responsible for all voluntomotoricity.[91] Control within the cortex involves simultaneous

activation of the spinal cord, brain stem, basal ganglia, and cerebellum.[91] Only after these lower centers are stimulated is when the muscles are activated.

Motor Cortex

The motor cortex is one of only two functional areas of the cerebral cortex. The motor cortex itself is divided into different areas: the primary motor cortex, the premotor cortex, and the supplementary motor area.[103, 104] The primary motor cortex is responsible for the control of fine voluntary movements.[103, 104] The neurons within this area are responsible for consciously controlling movements of the skeletal movements.[103, 104] The premotor area is more responsible for specific task movements.[103, 104] The premotor area together with the primary motor cortex and the supplementary motor area is responsible for constituting an overall system for coordinated muscle activity and development of motor skills. The premotor area has a role in the coordination and the execution of learned bilateral movements such as walking.[103, 104] As for the supplementary area, this area functions together with the premotor area to provide attitudinal movements.[103, 104] This area of the cortex is active immediately before the execution of movement, which suggests a role in the planning of movement.[103, 104]

Corticospinal Tract

When signals are originated within the motor cortex, they are transmitted directly to the spinal cord through the corticospinal tract (pyramidal tract) via the brain stem and the spinal cord.[104] The pyramidal tract is simply a descending tract from the motor cortex to the lower motor neurons.[104] There are two descending tracts from this area: the lateral and anterior corticospinal tracts, and the corticobulbar tracts. The corticospinal tract is the most important pathway for our concerns and will only discuss this pathway. The

corticospinal tracts descend from the primary motor area through the brain stem to the spinal cord.[104] Most of the pyramidal fibers cross to the opposite side and descend in the lateral corticospinal tracts of the cord terminating on the interneurons in the intermediate regions of the cord gray matter.[104] Corticospinal neurons of the motor cortex control the spinal reflexes. The pyramidal neurons not only connect within several motor neuron pools, but they provide direct monosynaptic connections with individual motor neurons, these may also be the same as neurons responsible for mediating spinal reflexes.[104] This allows the motor cortex to govern complex patterns of muscle activation through the control of automatic reflex behaviors organized at lower levels. [104]

Neurons of the motor cortex have peripheral receptive fields and are controlled by the somatic sensory system.[104] The somatic sensory system is responsible for providing information from the body, including skin, muscles and joints. Some neurons respond to tactile stimuli, others to joint rotation or compression, and others to stretch of muscles. This input-output association is responsible for the close relationship between the target muscle and the peripheral area that excites the neuron.[104] The motor cortex is excited by sensory information; it reacts with the appropriate course of motor action.

The highest level of control of motor activity is composed of the cortical motor areas together with the basal ganglia and cerebellum.[103] The cortical motor areas include the primary motor area, the premotor area and the supplementary motor area. Both the cerebellum and basal ganglia interact with the cortical motor areas to provide sufficient information for movement.

Motor Learning / Programming

A motor skill is a sequence of movements that are performed to achieve a particular goal with appropriate speed and accuracy.[104] The acquisition of a motor skill involves a learning period when the correct programs are developed in the central nervous system, and the activity in all the groups of muscles is gradually modified to produce the correct force and timing.[104] As a complex motor skill such as throwing a baseball is acquired; movement occurs slowly at first and is repeated many times relying heavily on feedback. Eventually the motor programs are executed with accuracy, unless significant changes in the environment occur during the progress.

Reflex Stabilization

The GH joint may be exposed to a perturbation and is consistently exposed to situations requiring quick transition from one direction to another during functional activity. These instability creating situations may be planned or unplanned, nonetheless each creates differences in the motor program that must be corrected. Reflex stabilization provided by dynamic stabilizers is of the utmost importance in order to maintain the humeral head within the glenoid. The musculotendinous envelope has been determined in a feline model.[93] Sensory branches of the axillary nerve consistently insert into the capsule. Direct stimulation of these afferent nerves resulted in reflex activation of the biceps, subscapularis, supraspinatus, infraspinatus, and acromio-deltoid muscles.[28]

Reflex arcs have been studied extensively in both the knee and ankle. Beard et al.[107] reported a significantly prolonged latency for hamstring contraction in anterior cruciate ligament deficient subjects. Pope et al.[108] studied reflex contraction of the sartorius and vastus medialis in response to a valgus stress on the medial collateral

ligament. They concluded that this arc was too slow to allow muscle contraction to play a protective role during downhill skiing. Lofvenberg et al.[109] found prolonged reflex firing of the peroneus longus and tibialis anterior in subjects with chronic lateral ankle instability. Lofvenberg et al.[109] and Beard et al.[107] studies showed longer muscle response times for joints with a history of injury when compared with normal patients. On the basis of these findings, we would expect the protective response times in a group of subjects with a history of traumatic instability to be longer than those with uninjured shoulders. A description of a reflex arc involving the GH joint was explained by Guanche et al.[110] utilizing a feline model. They indicated that stimulation of the anterior and the inferior axillary articular nerves elicited electromyographic activity in the biceps, subscapularis, supraspinatus, and infraspinatus muscles. They concluded that the existence of this reflex arc from mechanoreceptors within the GH capsule to muscles crossing the joint confirms and extends the concept of synergism between the static and dynamic restraints of the GH joint. The length of this reflex neural pathway (shoulder) is significantly shorter than the knee or ankle reflex neural pathways, and therefore muscle activation may occur in time to stabilize the GH joint prior to subluxation or dislocation occurs. In order to maintain functional joint stability the sensorimotor system incorporates all the afferent, efferent, and central integration and processing components. Integration of information essential to maintain stability is essential at multiple levels of processing.

Although visual and vestibular input contributes, the peripheral mechanoreceptors are the most important from a clinical orthopaedic perspective. (Figure 2.2)[18] The peripheral mechanoreceptors (pictured on the lower left) reside in the cutaneous,

muscular, joint and ligamentous tissues.[18] Afferent pathways (dotted lines) convey input to the 3 levels of motor control and associated areas such as the cerebellum.[18] Activation of motor neurons may occur in direct response to peripheral sensory input (reflexes) or from descending motor commands, both of which may be modulated or regulated by the associate areas (gray lines).[18] Efferent pathways from each of the motor control levels (solid lines) converge upon the alpha and gamma motor neurons located in the ventral aspects of the spinal cord.[18]

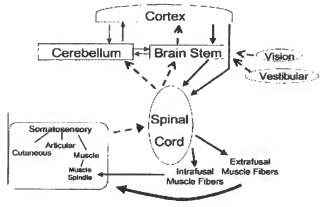

Figure 2.2. Sensorimotor pathways. [18]

Electrical stimulation of upper limb peripheral nerves[111] or the imposition of sudden load changes resulting in displacements of upper limb musculature[112, 113] results in a number of consistently timed EMG responses in humans. Tatton and Lee[114] indicated that responses are initiated at 10-12 msec, 32-35 msec and 45-60 msec in the primate wrist following the step-load change and have been termed the M1, M2 and M3 responses.[115, 116]

Tatton and Lee[114] later investigated these EMG responses in normal human subjects and indicated that the responses appeared to correspond to those observed in

monkeys, allowing for the longer conduction distances in man. The M1 and M2 responses varied in amplitude from subject to subject but were consistently present with latencies of 28-32 msec and 58-62 msec, respectively, while M3 responses were present in about 40% of subjects with latencies of 85-95 msec.

The latency of the M1 response suggests that it corresponds to the classic monosynaptic stretch reflex.[117] While the origin of the M1 response is generally agreed upon, the nature of the M2 and M3 response is still uncertain in the human. Tatton and Lee[114] have indicated that although the supraspinal pathways mediating the M2 and M3 responses are not firmly established, the M2 response appears to be mediated by a transcortical pathway involving the motor cortex[115, 116, 118], while the M3 response requires that the cerebellar peduncles are intact.[118] The M3 response is oftentimes referred to as voluntary muscle activity existing after the presence of the M2 peak.

The balance of evidence suggests that the initial component of the long-latency response (M2 response) evoked in the distal musculature of the upper limb is predominantly mediated by a transcortical pathway through the contralateral primary motor cortex.[119] This is supported by Tatton et al.[116] who indicated that after receiving a post central lesion the M2 response was eliminated in the primate. This finding is in accord with the physiological and anatomical evidence and strengthens the proposal that a transcortical pathway involving the post central cortex mediates the M2 response.

The GH joint is minimally constrained and relies significantly on neurologic influence and motor control for dynamic stabilization and is a productive site for reflex stabilization research. During the late cocking phase and the transition into the acceleration phase, it is essential that the internal rotators not only counteract the external

rotation force, but they must act in a timely manner. Reacting in a timely manner will allow more effective muscle function helping to combat excessive GH translation. Researchers have indicated changes among subjects with a history of injury compared with normal patients.[107, 109, 120] On the basis of these findings, we would expect the protective response times in a group of subjects with a history of traumatic instability to be longer than those of a normal group of subjects. No literature has been found that investigates the differences in muscle latencies and firing patterns in response to an external rotation perturbation in these subjects. This information may be important in establishing rehabilitation criteria and returning an athlete to optimal performance.

Auge and Morrison[27] and Brindle et al.[121] examined muscle activations and latencies in throwers and non-throwers. Each indicated a more quiescent reflex response for the trained overhead throwers vs. the non-throwing groups. Brindle et al.[121] examined muscle responses to an internal rotation perturbation, attempting to mirror the mechanism of the deceleration phase of throwing. They concluded that the more quiescent response leads to a greater time in the acceleration phase of throwing, enabling a greater pitching velocity before the rotator cuff muscles suddenly activate to decelerate the arm following ball release.[121] Auge and Morrison[27] on the other hand examined the infraspinatus during an imposed stretch. Throwers require an exponential amount of motion to help in maintaining extreme velocity demands during pitching. This is evidence of the altered muscle firing patterns and increased latencies incurred during throwing, leading to increases in humeral head translation. This is the basis of compromise between GH stability and function.

Glousman et al.[21] indicated that muscular activity is significantly decreased in those shoulders that are distinguished as unstable. The issue may not necessarily be the amount of force differences between those who are unstable and those who are normals, but in all likelihood it is the timing of the reflex response times during throwing. Latimer et al.[28] investigated the reflex response times in the GH joint among normal subjects to an anterior translation force. Subjects were supine with their shoulder in 90° of abduction and 90° of external rotation at the GH joint. Unknown to the subject a pulley system was activated resulting in an external rotation/ horizontal abduction perturbation to the shoulder. The data indicated that the anteriorly located muscles (pectoralis major, anterior deltoid, upper subscapularis, and long head of the biceps) all responded in 110 to 160 msec. They also indicated that anteriorly located muscles preceded the activation of the posteriorly located muscles. These latencies give us a view of the time demands that are available in the adjustment to the motor program of GH joint in normal subjects. The timing of these latencies is essential in maintaining dynamic stabilization during functional activity as well as during activities of daily living. Inhibition of internal rotation and protraction of the scapula accentuate the stresses on the anterior restraints thereby leading to increases in laxity and causing the continuum of the instability vs laxity paradigm. The reflex arc of the shoulder in response to an anterior translation force has not been studied in those with instability. Identifying the characteristic latencies and muscle firing patterns and differences among those with and without instabilities may assist clinicians in identifying the cause and effect of the instability vs. laxity question.

An individual with deficient capsuloligamentous structures, or one with poor strength and conditioning of the rotator cuff muscles, might theoretically be at greater risk of GH instability. A deficiency of the capsuloligamentous function may be due to the micro-traumatic nature of the activity causing a repetitive attenuation of the structures. Repetitive overhead motions with fatigue and failure of the dynamic stabilizers to contain the humeral head in the glenoid might produce excessive translation sufficient to plastically deform the capsuloligamentous structures.[122] If deformation occurs in the capsuloligamentous structures, the athlete is then able to achieve greater ranges of motion before the mechanoreceptors are deformed and provide proprioceptive guidance. Proprioceptive feedback from ligaments coordinates the protective effect of dynamic stabilizers during shoulder motion, and fatigue, capsular injury, or congenital laxity may interfere with this protective mechanism.[17, 58, 123, 124] An increase in laxity desensitizes the mechanoreceptors within the stabilizing constraints and allows for an increase in motion, meanwhile representing a delay in activation of dynamic restraint mechanisms. In addition, the congruent connections of the dynamic stabilizers within the capsule also play a role in stabilization and communication. When the dynamic stabilizers are activated creating a muscular contraction, tension is placed on the capsule, creating facilitation for stabilization. When fatigue or inadequate strength is noted within these structures, asynchronous muscle firing or improper muscle recruitment may be noted, resulting in deficiencies in stabilization leaving athletes to rely heavily upon the capsuloligamentous constraints for stabilization.

Many factors can be responsible for creating a shoulder instability environment in the athlete. Inability to control the humeral head within the glenoid may cause

capsuloligamentous attenuation. Capsuloligamentous attenuation may cause decreases in mechanoreceptor ability to activate appropriate structures to assist with stabilization. The paradigm of instability can be a continual battle if each of these causes of instability are not appropriately addressed. (Figure 2.3) It is the duty of current researchers and clinicians to continue their quest for the underlying dynamics of shoulder instability.

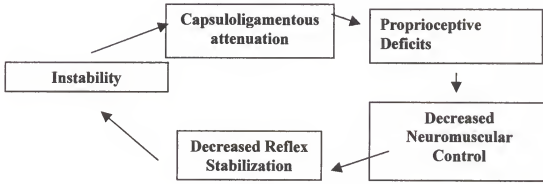

Figure 2.3. Paradigm of Instability

Conclusion

In summary, there is overwhelming evidence that there are deficiencies in the restraint mechanisms of the GH joint in the unstable shoulder. Deficiencies in the restraint provided by reflex stabilization mechanisms may result in susceptibility to shoulder injury and the promotion of the continual laxity vs. instability paradigm. The actions of reflex stabilization, which is influenced by the static and dynamic restraint mechanisms, allow for coordinated and efficient muscular function. A longer latency in response to an ER or IR perturbation of the GH joint in the unstable shoulder may aid and precipitate further instability and related pathologies. Testing the shoulder using the ER

and IR perturbation protocol on those with and without shoulder instability will be more functional and conclusive in the investigation of the underlying cause of instability. Therefore, an examination of the reflex stabilization amongst patients with and without stable shoulders is warranted. This study may benefit the clinician and rehabilitation professional when considering the utilization of reflex stabilization in regards to therapy for those with unstable shoulders.

CHAPTER 3
MATERIALS AND METHODS

Subjects

Subjects participating in this study included 10 males, ages 18-27. Subjects making up the control group (N = 5) had no history of shoulder dislocation or subluxation in their dominant shoulder and were asymptomatic from shoulder or upper arm pathology and otherwise normal at the time of the study. In addition, the control group was matched to the participating instability subjects by height, weight, age and dominant shoulder. Those that made up the instability group (N = 5) presented with a history of subluxation, or dislocation to their dominant shoulder, but presently were asymptomatic.

The sample size was selected based on the results from conducting a power analysis (f = 1.06, α = .05, 80 % power) utilizing data collected from previous studies that were similar in nature to the present project.[27, 28, 121] The power analysis revealed that 5 subjects per group (unstable, control) or a total of 10 subjects were needed to achieve appropriate power. Based on conventional effect sizes, this is considered a large effect, achieving high power even with a relatively small sample.[125]

Subjects were recruited from the College of Health and Human Performance, Department of Physical Therapy, and Department of Orthopaedics. Subjects who met the inclusionary criteria were approached by the primary investigator. Those individuals that maintained an interest in participating in the investigation were enrolled in the study. Satisfaction of the inclusionary and exclusionary requirements was determined by verbal

questioning and assessment by the principal investigator for all subjects. Those that were included in the control group were subjects that had never experienced a subluxation or dislocation episode in their dominant shoulder. To be included in the instability group, subjects must have had a previous history of shoulder dislocation or subluxation to their dominant shoulder. Subjects of either group were excluded from the study if they presented with any pain or discomfort in their dominant shoulder at the time of data collection. Additionally, all subjects signed an informed consent form, approved by The University of Florida Institutional Review Board (UFIRB-01), prior to assessments.

Instrumentation and Setting

Participants were asked to report on one occasion to the Physical Therapy Education Building at the University of Florida for approximately two hours of testing. Range of motion measurements (IR at 90, ER at 90, ER in neutral, extension, flexion) were collected on each subject using a hand held goniometer. For the purpose of this study a custom made perturbation set-up was developed to maintain positioning at 90° of GH abduction and a point equal to 90 % of each subjects' maximal GH external rotation. (Figure 3.1)

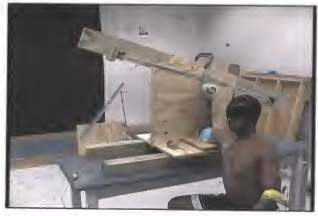

Figure 3.1. Subject positioning utilizing the perturbation device.

Attached to the set-ups arm stabilization device were two spring attachments, which applied tension to the subjects' shoulder in both internal and external rotation directions, providing the perturbation motion. These springs were attached to the stabilization device at a standardized tension in order to maintain consistency in velocity (≈570°/sec) between perturbation trials for each subject. In addition, the springs were situated in such a fashion, so that when subjects with differing ranges of motion were tested the angle of pull remained the same. The velocity of the perturbation was determined by utilizing a microswitch set-up that signaled the start of the movement and the end of the movement through a specified angular distance. The velocity calculation was completed with no limb attached to the perturbation arm. Upon activation, the springs provided an external or internal rotational displacement of the arm stabilization device. Surface EMG recordings from the infraspinatus were collected unilaterally using one 1.0-cm width silver/silver chloride electrodes embedded in an epoxy-mounted preamplifier system (35X), which maintained a constant inter-electrode distance of 1.25 cm. Intramuscular electrodes were inserted to collect EMG signals from the subscapularis, teres major, and supraspinatus. Fine wire electrodes (50 micron) with two 2 to 3 mm-bared tips were placed into the above targeted muscles through a 25-guage hypodermic needle used as a cannula. All fine wire electrodes were steam autoclaved in the Department of Orthopedics, University of Florida before insertion; in addition, the fine wires were discarded after each use. A new wire was cut for each muscle and subject being tested. The EMG signal was band pass filtered (40-4000 Hz) and rectified on line. The amplifier gain was set to reproduce a signal suitable for visual interpretation. The processed signal was sampled on-line for 300 ms at a sampling rate of 2000 Hz.

Measurements

The displacement provided by the perturbation device set-up had the effect of externally or internally rotating, and stressing the dominant GH joint. Muscle latencies were investigated in response to the ER and IR perturbation of the dominant shoulder of both groups (Control, Instability). Muscle latencies (msec) were determined by identifying the time from the onset of the perturbation to the onset of muscle activity. Onset was detected by the use of a microswitch set-up that marked the EMG when the movement arm was initially displaced. Latencies were quantitatively identified by measuring the baseline muscle activity, which was noted by the rectified mean muscle activity 300 msec prior to the onset of the perturbation. In order to identify the muscle onset, a 3 standard deviation method was employed.[121] A 3 standard deviation activation threshold was used to minimize the probability of incorrectly describing an inactive muscle as being active. The muscle was identified as having reached activation when the muscle activity after perturbation reached a point that equaled 3 times the rectified standard deviation of the mean baseline activity. The baseline activity and calculation of the standard deviation method for muscle onset determination was completed for each muscle during each subsequent trial.

Procedures

After obtaining the subject's informed consent, demographic (height, weight, age, dominant shoulder) and range of motion measurements (IR in neutral, IR at 90/90, ER in neutral, ER at 90/90) were collected. In addition, a point of 90 % of the maximum ER in 90° abduction was calculated, for use as the position of the GH joint upon perturbation. Subjects were placed in a stable seated position with their shoulder and arm placed in 90°

of abduction and to a position that equaled 90 % of the subject's maximum external rotation. Once the subjects were placed and constrained to the perturbation device, headphones and a blindfold were placed onto the subjects. The purpose of these items was to achieve an unexpected perturbation during trials. Subjects were then familiarized with the manipulandum set-up and function. In order to familiarize the subject, perturbation trials in the ER and IR directions were administered until the subject felt comfortable with the tasks. Subjects ER and IR were measured earlier to identify the normal pain free range, in order to maximize subject comfort during the session. Testing was only carried out within this protected range. Range of motion stops were placed within the perturbation device to ensure that the ER or IR perturbation did not exceed the previously mentioned pain free range of motion. As the perturbation was applied to the GH joint, the subject was asked to resist the movement and keep the shoulder from moving in the direction of the perturbation.

Once the subjects felt comfortable with the perturbation device, they were cleansed and prepared for the placement of the fine wire and surface electrodes. From this point on, an aseptic approach was utilized. For those muscles (subscapularis, teres major, and supraspinatus) being examined, utilizing fine wire techniques, a single needle technique as described by Basmajian and Deluca[126] was used. Insertion of the fine wire electrodes was carried out by the investigators. Electrode placement of the fine wires was confirmed by selective muscle testing for each muscle, as described by Wilk and Andrews.[127] If correct placement of the wire electrode was in doubt, then new wires were either reinserted or the subject was given the option to withdraw from the study. (Appendix A) Surface electrodes were also attached to the infraspinatus for investigation.

Once electrode placement was verified, the subjects were placed into the perturbation device as previously mentioned. Subjects were asked to relax and informed that testing was about to begin, they were also reminded that once they felt the perturbation they were requested to resist the movement created by either the IR or ER perturbation. At a time unknown to the subject and at varying resting durations, the perturbation was administered to the GH joint in an ER or IR direction. All movements were controlled for velocity by the use of the same perturbation spring setup with equal tension during all tests and verified by a micro switch set-up. The velocity of the perturbation device was examined after each subject to ensure equal perturbation velocity in each direction between each subject. The range of motion through each of the perturbations was also controlled by beginning the perturbation at a point that equaled 90% of the subjects max ER and limited the perturbation at a position that did not exceed the subjects maximum pain free ROM. This was confirmed by the use of mechanical stops placed at the point in the range where they had been identified as reaching their maximum range of motion in the ER and IR direction, without discomfort. EMG activity was collected during each of the ER and IR perturbation trials. Subsequent trials were not initiated until the subject advised that they were fully recovered and prepared to carry out the task. The order of perturbation direction was randomized using a coin flip. The subjects were instructed and encouraged to perform every repetition at maximal effort and in as little time as possible. Testing was completed when at least 7 of the trials exhibited a quite baseline and appropriate muscle onset. An appropriate muscle onset and EMG activity was characterized by a signal that presented with a clear increase in activity and did not exist with noise up to the onset. Below is an example of an acceptable and unacceptable trial. (Figure 3.2, Figure 3.3)

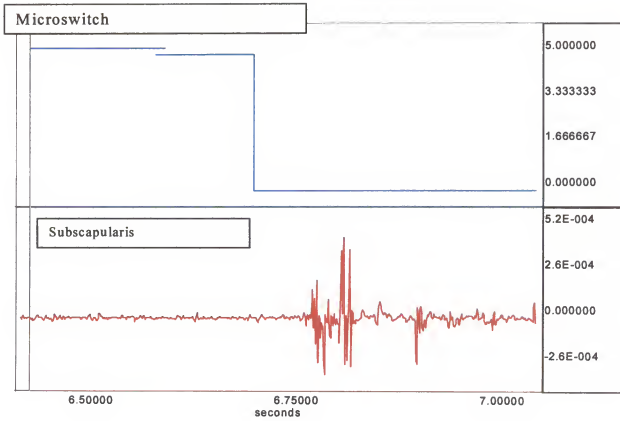

Figure 3.2. Acceptable reflex latency onset.

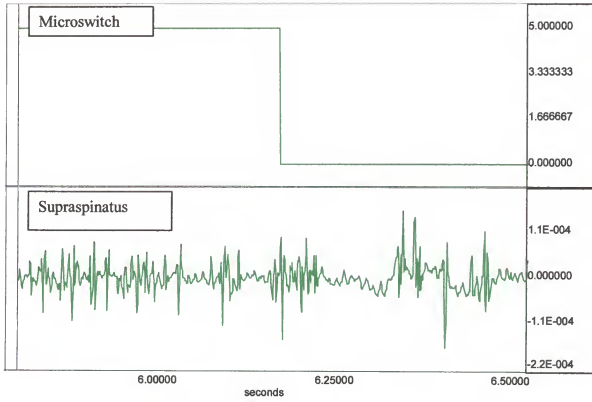

Figure 3.3. Unacceptable reflex latency onset.

Design and Analyses

Comparisons were made between subjects with instability versus those with stable shoulders (control). The latencies (msec) from the unstable group were compared to the latencies noted in the dominant shoulder of the control group.

A three way mixed model analysis of variance (ANOVA) was performed. The between subjects factor was group, which included two levels, the control group and the unstable group. The within subjects factors were the muscles investigated and movement. The muscles investigated include four levels (subscapularis, teres major, supraspinatus, and infraspinatus); and movement had two levels; internal rotation, and external rotation. The dependent variable measured was muscle latency (msec). An a priori α level of significance was set at $p < 0.05$ for all comparisons. Significant differences were followed by Tukey HSD post-hoc analysis. The SPSS for Windows 9.0 (SPSS Inc., Chicago, IL) was used for statistical analyses.

CHAPTER 4
RESULTS

The purpose of this study was to determine if there were differences in the reflex stabilization (latencies) of the shoulder musculature (subscapularis, teres major, infraspinatus, and supraspinatus) in stable and unstable shoulders in response to an external (ER) or internal (IR) rotational perturbation. The means of the muscle latencies in response to the ER and IR perturbation trials used in data analysis are found in Appendix C. ANOVA tables are placed in Appendix D and the Tukey HSD Post-Hoc analyses calculation of critical values are located in Appendix E.

Subject Demographics

Subjects participating in this study included 10 males, ages 18-27. Subjects making up the control group (N = 5) had no history of shoulder dislocation or subluxation in their dominant shoulder and were asymptomatic from shoulder or upper arm pathology and otherwise normal at the time of the study. In addition, the control group was matched to the participating instability subjects by height, weight, age and dominant shoulder. No significant differences were noted between groups with the dependent T-test comparison of the means for age, height and weight. (Appendix F) Those that made up the instability group (N = 5) presented with a history of subluxation, or dislocation to their dominant shoulder, but were asymptomatic at the time of study. All testing procedures were performed on the dominant limb (8 right, 2 left). Demographic statistics of both groups (instability, control) are illustrated in Table 4.1.

Table 4.1. Subject demographics (Mean ± SD).

Group	Age	Weight	Height
Unstable	22 ± 3.08 yrs.	87.09 ± 13.37 kg.	177.80 ± 3.58 cm.
Control	21.6 ± 2.07 yrs.	85.73 ± 12.56 kg.	177.80 ± 3.58 cm.

Statistical Analysis

Comparisons were made between subjects with instability and those with stable shoulders (control). The latencies (msec) from the dominant shoulder of the unstable group were compared to the latencies noted in the dominant shoulder of the control group. These latencies were compared in response to both internal and external rotational perturbations.

One, three way mixed model analysis of variance (ANOVA) with repeated measures was performed for reflex latency. The between subjects factor was group, which included two levels, the control group and the unstable group. The within subjects factors were the muscles investigated and movement. The muscles investigated included four levels (subscapularis, teres major, supraspinatus, and infraspinatus); and movement had two levels; internal rotation and external rotation. The dependent variable measured was reflex latency. An a priori α level of significance was set at $p < 0.05$ for all comparisons. Significant differences were followed by Tukey HSD post-hoc analysis. The SPSS for Windows 9.0 (SPSS Inc., Chicago, IL) was used for statistical analysis. This analysis produced six tests of significance: a test for the main effects of movement, muscle, and group and tests for two-way interactions between movement and group, muscle and group, and movement and muscle, and a test for a three-way interaction between movement and muscle and group.

ANOVA--Reflex Latency

Muscle reflex latencies were extracted from the EMG recording (supraspinatus, infraspinatus, subscapularis, teres major) in response to a perturbation to the dominant shoulder in either the internal rotation or external rotation direction between the stable (control) and unstable groups. The ANOVA revealed significant differences in the

movement, muscle and group main effects, the movement by group interaction, the muscle by group interaction, the movement by muscle interaction and the movement by muscle by group interaction. (Appendix D)

Group Main Effect

A significant main effect for group [$F(1,8) = 63.434$, $p < .001$] was noted. It must be stressed that this main effect pools reflex latency times from both movements (IR, ER) and all muscles (supraspinatus, infraspinatus, subscapularis, teres major). The latency values for the control group (52.654 ± 2.632) were significantly shorter ($p < .001$) than the reflex latencies for the unstable group (82.302 ± 2.632). (Figure 4.1)

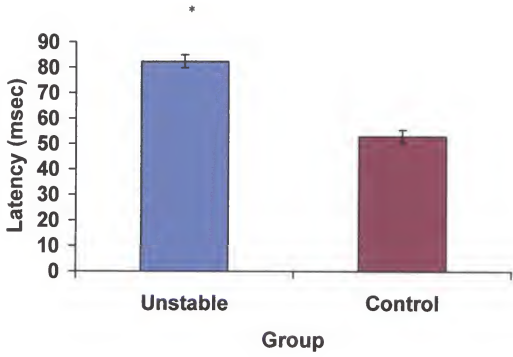

Figure 4.1. Significant main effect for group.
* indicates a significant difference between unstable and control groups.

Movement Main Effect

A significant main effect for movement [$F(1,8) = 32.938$, $p < .001$] was noted. It is important to remember that this main effect pools reflex latency times from both groups (control and unstable) and all muscles (supraspinatus, infraspinatus, subscapularis, teres major). The latency values during internal rotation (60.304 ± 1.807 msec) were significantly shorter ($p < .001$) than the latency values during the external rotation perturbation (76.652 ± 2.605 msec). (Figure 4.2)

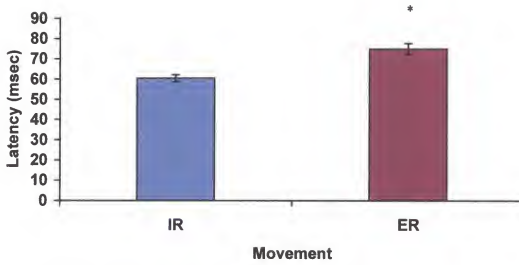

Figure 4.2. Significant main effect for movement.
* indicates significant difference between ER and IR.

Movement by Group Interaction

A significant movement by group interaction [$F(1,8) = 37.712$, $p < .001$] was noted. The Tukey post hoc analysis determined that differences of ≥ 11.32 msec were needed to indicate significant differences. Upon close examination of the appropriate means in the group comparison, the control group internal rotation (CON IR; 53.157 ± 2.556) and control group ER (CON ER; 52.152 ± 3.684) had significantly faster responses than the unstable internal rotation (UN IR; 67.452 ± 2.556) and unstable external rotation (UN ER; 97.152 ± 3.684) groups. In addition, significantly shorter reflex latencies were observed when comparing IR to ER within the UN group. (Figure 4.3)

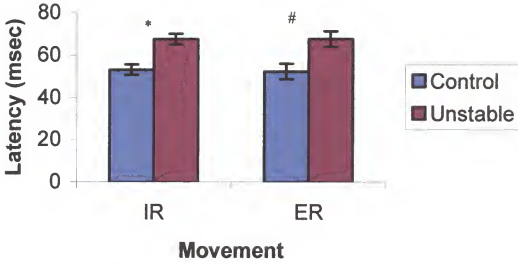

Figure 4.3. Significant movement by group interaction.
* indicates significant difference between CON IR and UN IR.
# indicates significant difference between CON ER and UN ER.

Muscle main effect

A significant main effect for muscle [$F(3, 24) = 13.256$, $p < .001$] was noted. The muscle main effect pools the latency values from each group (CON, UN) and movement (ER, IR). The latency values for the infraspinatus muscle (50.580 ± 2.349) were significantly shorter ($p < .001$) that those noted in the subscapularis (78.016 ± 4.899), supraspinatus (70.719 ± 2.857), and teres major (70.597 ± 2.760). (Figure 4.4)

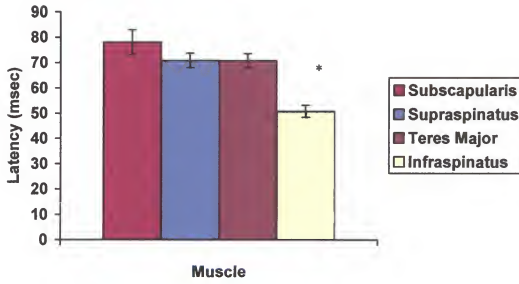

Figure 4.4. Significant muscle main effect.
* indicates that the infraspinatus has a significantly shorter latency than the subscapularis, supraspinatus and teres major.

Muscle by Group Interaction

A significant muscle by group interaction [$F(3,24) = 6.052$, $p < .004$] was noted. The Tukey post hoc analysis indicated that a value of ≥ 21.43 msec was needed to indicate significant differences. Upon close evaluation of the means for the group comparison, the control subscapularis (CON SUB; 55.181 ± 6.923), control supraspinatus (CON SUP; 56.496 ± 4.040) and control teres major (CON MAJ; 52.468 ± 3.904) all exhibited significantly faster reflex latencies than the unstable subscapularis (UN SUB; 100.851 ± 6.928), unstable supraspinatus (UN SUP; 84.943 ± 4.040), and unstable teres major (UN MAJ; 88.726 ± 3.904). In addition, significantly faster reflex latencies were observed in the infraspinatus (UN INF; 54.688 ± 3.322) in comparison to the subscapularis (UN SUB; 100.851 ± 6.928), supraspinatus (UN SUP; 84.943 ± 4.040) and teres major (UN MAJ; 88.726 ± 3.904) within the unstable group. (Figure 4.5)

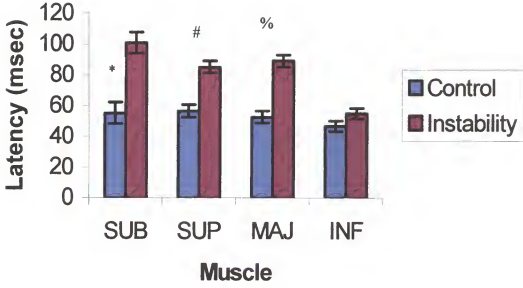

Figure 4.5. Significant muscle by group interaction.
* indicates a significant difference between CON SUB and UN SUB.
# indicates a significant difference between CON SUP and UN SUP.
% indicates a significant difference between CON MAJ and UN MAJ.

Movement by Muscle interaction

A significant movement by muscle interaction [F (3,24) = 3.676, $p < .03$] was noted. The Tukey post hoc analysis indicated that a value of $\geq$14.82 msec was needed to indicate significant differences. Upon close evaluation of the means for the movement comparison, the SUB IR (66.076 ± 4.369) and SUP IR (61.875 ± 2.919) had significantly shorter latencies than the SUB ER (89.956 ± 6.496) and SUP ER (79.564 ± 3.910). In the muscle comparison a significantly shorter latency was indicated for the INF IR (44.496 ± 2.164) than the SUB IR (66.076 ± 4.369) and SUP IR (61.875 ± 2.919). While the INF ER (56.664 ± 4.112) exhibited a significantly faster latency than the MAJ ER (72.424 ± 2.960) and SUP ER (79.564 ± 3.910). (Figure 4.6)

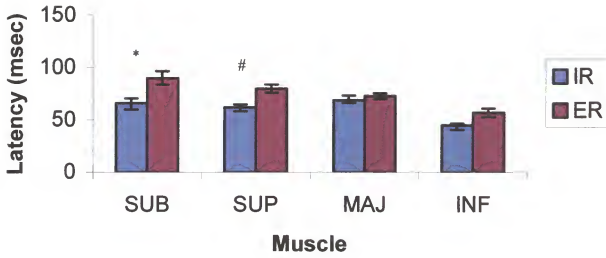

Figure 4.6. Significant movement by muscle interaction.
* indicates significant difference between SUB IR and SUB ER.
# indicates significant difference between SUP IR and SUP ER.

Group by Movement by Muscle Interaction

A significant group by movement by muscle interaction [F (3,24) = 6.439, p < .003] was noted. The Tukey post hoc analysis indicated that a value of ≥22.84 msec was needed to indicate significant differences. The means for the muscle comparison for UN IR the INF (48.452 ± 3.060) had a significantly shorter latency than the SUB (78.008 ± 6.179) and MAJ (80.482 ± 5.954). For the muscle comparison for the UN ER the SUB (124.694 ± 9.187) indicated a significantly longer latency than the MAJ (96.97 ± 4.186) and the INF (60.924 ± 5.815). In the group comparison for IR the CON SUB (55.144 ± 6.179) had significantly shorter latency times than the UN SUB (78.008 ± 6.179). The CON MAJ (57.058 ± 5.954) was significantly shorter than the UN MAJ (80.482 ± 5.954), and UN MAJ (80.482 ± 5.954). (Figure 4.7) In the group comparison for ER the CON SUB (55.217 ± 9.187), CON SUP (53.108 ± 5.529), CON MAJ (47.878 ± 4.186), and CON INF (52.405 ± 5.815) all had significantly shorter latencies than the UN SUB (124.694 ± 9.187), UN SUP (106.020 ± 5.529) and UN MAJ (96.97 ± 4.186). (Figure 4.8) According to the Tukey post hoc analysis for the movement comparison the UN SUB IR (78.008 ± 6.179), had significantly shorter latencies than the UN SUB ER (124.694 ± 9.187). While the UN SUP IR (63.866 ± 4.127) and UN INF IR (48.452 ± 3.060) had shorter latencies than the UN SUB ER (124. 694 ± 9.187), UN SUP ER (106.020 ± 5.529), and UN MAJ ER (96.812 ± 4.480).

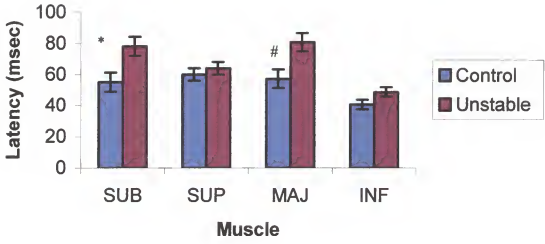

Figure 4.7. Significant group by movement by muscle interaction for IR.
* indicates significant differences between CON SUB and UN SUB for IR perturbation.
# indicates significant differences between CON MAJ and UN MAJ for IR

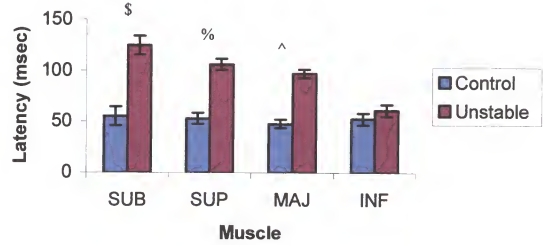

Figure 4.8. Significant group by movement by muscle interaction for ER.
$ indicates significant differences between CON SUB and UN SUB for ER perturbation.
% indicates significant differences between CON SUP and UN SUP for ER perturbation.
^ indicates significant differences between CON MAJ and UN MAJ for ER perturbation.

CHAPTER 5
DISCUSSION

The purpose of this study was to determine the timing (latencies) of muscular firing in normal human subjects versus those with shoulder instabilities of the GH joint in response to an internal and external rotation perturbation to the dominant shoulder. The dependent variable chosen to assess muscular firing was a measure of reflex stabilization, described as muscle latency (msec). This variable was assessed during both an IR and ER perturbation with the subject positioned at 90° of GH abduction and a point equal to 90 % of their maximal GH external rotation. Through an exhaustive literature review, no other study was found to investigate reflex muscle latencies in response to an ER and IR perturbation between subjects with and without shoulder instability. Other studies[28, 121] investigated latencies of normal and athletic subjects but did not incorporate both perturbation directions or involve the "pathologic" population. The present study takes a functional and realistic approach to investigating reflex latencies.

Previous authors[107-109] have identified prolonged reflex stabilization when investigating various injured populations. Similarly, prolonged reflex stabilization in the unstable shoulder was noted in the findings of our investigation. Deficits in reflex stabilization have been attributed to damaged or deafferentated mechanoreceptors, which normally contribute to joint proprioception and reflexive stabilization. Lephart et al.[17] examined shoulder proprioception in normal, unstable, and surgically repaired individuals. They noted that subjects with anterior shoulder instability demonstrated

significant deficits in proprioception as measured by repositioning of a passive movement and threshold to detection of passive motion, when compared to normal individuals. It was hypothesized that acute and chronic injury to the GH capsule and ligaments interferes with mechanoreceptor function and joint proprioception. Anterior stability of the GH joint is accomplished primarily by the restraints of the anterior portions of the GH joint (capsule, subscapularis, teres major, glenoid labrum). These primary restraints may become attenuated and lax in response to injury or repetitive loading, allowing asymmetrical increases in range of motion that may potentially precipitate instability in throwing athletes. This may result in asymmetrical increases in external rotation of the humerus in the power phases of the pitch thus perpetuating and/or producing further humeral translation and instability. In the complex shoulder, distention of the joint capsule or injury to dynamic stabilizers may result in a delayed reaction to preparatory and reactive muscle activation.[22] This phenomenon represents inadequacies in the neuromuscular function of the shoulder, and is reason for the importance of identifying a precipitating cause of the shoulder instability paradigm.

Capsular distention may be the result of injury in response to dislocation, subluxation or micro-traumatic throwing episodes. Moseley and Overgaard[44] showed that laxity of the subscapularis of varying degrees and occasional attenuation of the tendon would accompany the pathological changes in the capsule resulting from trauma. Muscular function has been found to be affected in those subjects with a history of shoulder injury.[20-22] Our investigation supports the work of Moseley and Overgaard.[44] We identified that a significant difference existed between reflex latencies in the control and instability (longer latencies) groups in response to both the IR and ER perturbation

trials. Decrements in proprioceptive communication due to structural damage may precipitate instability pathologies and result in decreased stabilization during functional activity.

Our investigation proved to support the first hypothesis that longer latencies in the instability group would be evident in response to the ER perturbation as compared to the control group. A significantly shorter latency for the control group supraspinatus, subscapularis and teres major was identified in comparison to their respective muscle in the unstable group. In addition, the second hypothesis that longer latencies in the instability group would be evident in response to the IR perturbation as compared to the control group was also supported with this investigation. A significantly shorter latency for the control group subscapularis and teres major was identified in comparison to their respective muscle in the unstable group. This indicates that after injury to the anterior stabilizing structures, delayed reflexive muscle activation results, which affects stabilization adversely. These results can be supported by others,[107-109] who investigated various joints within the body and identified prolonged reflex latencies between injured and healthy populations.

Lofvenberg et al.[109] examined patients with functional ankle instability and normals. They subjected patients with symptoms of chronic lateral instability of the ankle to sudden angular displacement. The reaction times in the peroneus longus and the tibialis anterior muscles were recorded and compared with the control ankles. Their investigation noted a significantly longer ipsilateral reaction time in the patients with functional ankle instability compared to the controls. They concluded that delayed proprioceptive response to sudden angular displacement of the ankle may predispose an

individual to, or be a cause of chronic lateral instability of the ankle. Perhaps, regardless of the location of the deficit, similar changes in the reflex neural pathway exist in response to attenuation resulting from injury, which creates decrements in the stabilization characteristics of injured individuals.

In addition to the findings of the unstable group response to the ER and IR perturbation, the unstable IR latencies for the subscapularis and supraspinatus were significantly shorter than their respective unstable ER latencies. This may give support to the communication amongst the static and dynamic restraint mechanisms which occurs during the acquisition of restraint. Although an attenuation of the anterior stabilization structures may be evident in the instability group,[17, 128] when the IR perturbation is imposed upon the GH joint it is evident that a shorter latency is noted than when an ER perturbation is applied. The musculature of the GH joint (dynamic restraint mechanism) and the passive restraint mechanisms create a holistic collection in the development of the capsule. As the cuff muscles contract simultaneously, forces generated in their tendinous insertions apply tension to the joint capsule.[81, 82] This increased capsular tension aids in drawing the humeral head into the glenoid fossa, supplementing joint stability.[82] When the IR perturbation is applied to the GH joint the infraspinatus is the primary restraint against the perturbation as compared to the other musculature investigated. As the infraspinatus is activated to restrict IR, it dynamizes the capsule signaling the other musculature to fire. The response that occurs from IR perturbation in the infraspinatus is elicited in less time than when acting in response to the ER perturbation. The elongated time for reflex stabilization to occur in response to ER perturbation may be due to capsular attenuation. A greater amount of attenuation allows

further physiological range of motion to occur prior to dynamization and activation of the appropriate stabilizing mechanisms. Kronberg et al.[22] identified that the infraspinatus plays an important role in shoulder stability, reducing strain of the anterior structures of the GH joint because of the infraspinatus' capacity to pull the humeral head posteriorly during external rotation.[79] This demonstrates support for our finding that the infraspinatus remained active in response to both perturbation forces, and does so in less time than the other stabilizing structures (teres major, supraspinatus and subscapularis). Cain et al.,[79] in cadaveric experiments, demonstrated that maximal contraction of the posterior rotator cuff muscles reduced anterior ligamentous strain. Blasier et al.[60] biomechanically studied the role of the rotator cuff and concluded that the muscles of the entire cuff contributed equally to stability. If tension on any one of the stabilizing muscles was reduced, the entire rotator cuff was omitted or significantly reduced, creating a substantial reduction in anterior joint stability compared with when tension was applied to all components.[6]

Another potential cause leading to a reduction in anterior joint stability was documented by Hancock and Hawkins[128]. They identified a necessity of assessment of shoulder instability as including the investigation of the posterior capsule for inconsistencies, such as tightness. Harryman et al.[88] showed that posterior capsule tightness can contribute to anterior subluxation and obligatory anterior superior translation of the humeral head. Even though the latency of the infraspinatus of the unstable group in response to the ER perturbation was not significantly different in comparison to its reaction to the IR perturbation, it did in fact have a shorter latency than the subscapularis, supraspinatus and teres major. The delayed firing of the subscapularis,

supraspinatus and teres major and normal firing of the infraspinatus may precipitate instability episodes.

We have discussed the reasoning behind the significant differences in the muscle and group comparisons identified with this investigation. In support for Hancock and Hawkins,[128] it is equally important to discuss the reasoning behind the significant movement comparisons. Latency differences in the subscapularis, and the supraspinatus in the IR (shorter) perturbation in comparison to the ER perturbation trials existed for the unstable group. Congenitally tighter and more "stiff" posterior capsule structures may account for the shorter latencies. Although analysis of range of motion and stiffness data on the current subjects was not carried out, they all presented with an overall decrease in IR in comparison to their ER range of motion. Typically we found that with a decrease in the IR measurement, a reciprocal increase in the ER motion existed, creating an equivalent envelope of motion for each subject. With this it could be hypothesized that the posterior capsule of the GH joint may involve greater stiffness than that which was exhibited intrinsic to the anterior capsule. If this held true, despite membership within the unstable group a shorter latency would result from perturbation in either direction due to the earlier notification of the restraint mechanisms in the posterior capsule. The earlier communication of the posterior capsule initially due to changes in motion in response to the IR perturbation then may allow earlier communication to the other stabilizing mechanisms by "dynamization" of the capsule.

Muscle stiffness is a mechanism responsible for functional joint stability and also is an integral portion of preparatory muscle contraction. As a result of pre-activation, muscle stiffness is believed to increase. McNair[129] defined muscle stiffness as the ratio of

change in force per change in length. This increased muscle stiffness resists stretching episodes both static and dynamic, heightens muscle spindle sensitivity, and reduces the electromechanical delay involved in reflexive stabilization.[129, 130] The inherent stiffness of the posterior capsule may promote increased stabilization when stressed regardless of instability. In essence, a stiffer muscle produces a stiffer, more functionally stable joint.

In order for the shoulder muscles to prevent instability by concavity compression, they must respond before joint subluxation occurs. The reflex response times after the application of a perturbation force to the shoulder were as expected possessing significant differences between unstable and controls. Eventhough, reflex response times are significantly longer between control and instability patients, it was not the aim of this investigation to determine whether or not these reflex responses are in fact significant enough in amplitude to overcome excessive stresses placed upon the GH jt.

The strength of this study is that the experimental model is a realistic approximation of the actual mechanism of a traumatic and repetitive force that would result in pathologic anterior translation of the humeral head. Other studies in the literature report reflex response times differing from this study; however, it is difficult to compare these studies, because of the wide variation in methods that exist.

Gaunche et al.[110], reported the timing of the reflex response to direct stimulation of afferent nerves in a feline model as approximately 3 msec. This response has been postulated to be fast enough to aid in maintaining joint stability. However in the situation of traumatic anterior translation force to the humerus, the nerve would not be receiving direct stimulation. Rather it would rely on the sequence of events leading to eventual activation to maintain stabilization.

Latimer et al.[28] reported reflex response times of 110-160 msec. These times are significantly longer than what was found in the present investigation. Latimer et al.[28] investigated the reflex response times in response to an ER perturbation force in normal shoulders, this is why the surprise of there results in comparison to ours. Differences in testing methods may contribute to the differences in reaction times that exist between the present study and ours. The method utilized for perturbation in the Latimer et al.[28] study was that of dropping a weight attached to a supporting rope. Gravity acceleration of the hanging weight upon release caused shoulder motion in the ER direction that stretched the capsule. They attempted to minimize "slack" in the system by placing the arm in maximal external rotation while maintaining muscular relaxation. There are obvious implications related to subject positioning in this investigation. Not all subjects represent a standardized perturbation position and therefore capsular tension prior to the weight application is variable, thereby creating differences in reflex response. Some subjects may have not been in maximum external rotation prior to perturbation, which would result in lengthened time for capsular stretching prior to muscle activation. In our study, subjects were placed in a standardized position that maintained their GH joint in a position of 90° GH abduction at 90% of maximum ER. In addition, subjects were placed into the perturbation device in a sitting position in a gravity independent position unlike that of Latimer et al[28]. Upon release of the micro-switch set up, the perturbation was activated ($\approx$ 570°/sec), there was no waiting for "slack" to be taken out prior to perturbation onset, as was seen with the Latimer et al.[28] investigation. These simple position differences and the nature of our perturbation device utilizing a spring setup may solely contribute to the timing differences between studies.

Pope et al.[108] suggested that knee ligament injury occurs at approximately 73 msec after ski engagement of downhill skiers. Their investigation identified that muscle reflexive activity wasn't evident until 128 msec. They suggested that the timing of the muscle activation is too long to prevent injury. We cannot speculate on whether or not the latencies found in the present study occur in time to prevent injury, but it can be hypothesized that those without a history of instability flirt with the extremes in muscle stabilization before deformation occurs in the affected structures. And those with shoulder instability are at a considerable risk for further or recurrent damage to the GH joint due to significantly longer latencies. If the musculature of those with shoulder instability reacts in such a delayed mechanism this can only create, precipitate and encourage the activation of the shoulder instability paradigm.

Auge et al.[27] identified similar response times to that noted in the present study, in their normal and multidirectional instability subject populations. The purpose of the contrasted study was to identify differences in the infraspinatus reflex response between groups of normal, athletic, and multidirectionally unstable shoulders. Subjects were asked to resist an internal rotation torque provided by an isokinetic dynamometer. In a sequential mode an isometric internal rotation torque was applied, followed by an increase in torque. This increase in torque resulted in internal rotation through a 30° arc at 150°/sec. Subjects were trained to maintain this specified limb position against a steady torque on which the additional torque was superimposed. The authors indicated that shoulders with multidirectional instability exhibited a more-prominent spinal stretch reflex response than normal shoulders, but did not indicate a difference in the timing between the two groups. This was a consistent finding with the present investigation,

keeping in mind that Auge et al.[27] tested only the infraspinatus. Due to the differences in the application of the aforementioned perturbation, the results may have separate implications. This gives further evidence that the infraspinatus is not affected between the normal and unstable groups in response to these varying methods of perturbation application. In addition the instability groups may present with a healthy infraspinatus even after bouts of injury, re-emphasizing the possible interpretation of stiffness and infraspinatus firing which in addition to longer latencies in the other stabilizing muscles around the shoulder lead to anterior instability.

Other studies[121] have utilized equivalent methods for determining the reflex latencies as the present study, although techniques for reflex latency determination vary greatly.[28, 107-109] The differences in the determination of the reflex latencies investigated in previous studies may be the sole reason for such differences in the timing regardless of location of the tested limb. The present investigation utilized a 3 standard deviation method; in which the muscle was described to be active once the activity after perturbation reached a point 3 times that of the pre perturbation mean activity.[128] It is essential to maintain critical requirements for data extraction of the reflex latencies. Identifying latencies among multiple joints in the body is important, but some variation will exist due to the neurological framework (distance) that may exist from different portions about the body.

The goal of this investigation was to determine the timing of musculature responsible for maintaining GH joint stability between those with a history of shoulder instability and normals in response to an ER and IR perturbation. The findings of this investigation support the previous data of other limbs.[107-109] Contemporary authors

suggest that a disruption to the mechanoreceptors will alter the coordinated muscle firing sequences of the force couple, which is essential to dynamic restraint and neuromuscular control. Asynchronous firing of the humeral and scapular muscles can lead to repetitive microtrauma and functional instability. In addition, these deficits alter joint reaction time to stress, therefore decreasing involuntary muscle activity that is necessary for dynamic restraint. The findings of this study support the notion of delayed reflexive muscular activity after injury and have obvious implications for the injured population active in sport activities.

Conclusions

Several conclusions have been drawn from the results of this investigation:

1. In terms of muscle latencies (msec); the unstable group possessed significantly longer latencies than the control group for both internal and external rotation perturbations forces. This has important implications for shoulder stability.

2. The injured (instability) GH joint in response to an IR will result in delayed activation of the subscapularis and teres major. This gives further evidence of the damage that may occur in response to a subluxation or dislocation episode.

3. When the GH joint is resisting an ER perturbation the subscapularis, supraspinatus, and teres major of the unstable shoulder will take longer to react and provide joint stabilization. This has implications for stability as the primary restraints against external rotation about the GH joint are incapacitated when they may be most crucial in the development of stability.

4. The subscapularis and supraspinatus in response to an IR perturbation are much more capable of creating a stabilization reflex than when these muscles are required to assist in stabilization during an ER perturbation. This may fuel support for the notion of "dynamization" of the capsule. This notion serves as support for the synergistic action of the active and passive restraint mechanisms, which are all encompassing of the GH joint capsule.

Summary

The aim of this investigation was to assess the differences in reflex stabilization in response to an IR and ER rotational perturbation in subjects with shoulder instability and those without. A total of 10 subjects in two groups (instability, control) were asked to resist both ER and IR rotational perturbations to their maximal effort. Shoulder instability was defined as a subject having a history of subluxation or dislocation to the dominant shoulder. Muscle latency was tested by the use of both surface (infraspinatus) and fine wire (subscapularis, supraspinatus, teres major) electromyography (EMG). During the testing sessions a micro-switch setup was initiated to indicate the start of each perturbation (IR or ER). The time from which perturbation was initiated to the time when significant muscle activation was noted, designated the reflex latency. Specifically, the measures of reflex latency that were recorded in this investigation were for each muscle involved for each subsequent trial or both ER and IR rotational perturbations.

The results of this study indicate that the unstable group possessed significantly longer reflex latencies than the control group for both motions. The data also indicated specific muscles that accounted for the deficit in timing of these latencies in the unstable groups. In addition, the structures that were noted as being involved in response to the instability episodes had a much shorter reflex latency when responding to an IR perturbation than the ER perturbations.

Given the results of this study, it is safe to assume that significant decrements in muscle latency exist between those with shoulder instability and those without. In addition, it may be justifiable to make mention of the dynamization that occurs within the GH joint capsule to maintain functional stability. In conclusion, when an athlete with

shoulder instability is presented, the practitioner must identify these areas of deficiency and implement a rehabilitative protocol aimed at decreasing the reflex latencies and maximizing GH joint stabilization.

Implications for Future Research

While this study has provided some interesting and informative insight into functional stability of the GH joint. This investigation is only a precursor to a vast expansion into the scientific body. The present study being the only of its kind has obvious implications for shoulder instability research in the future. Below are several unresolved issues resulting from this investigation.

1. This question of what occurs first is an obvious dilemma and may fuel further research. The cyclical notion of shoulder instability recreates the question: "Does injury cause increased reflex latency, or does increases in reflex latency cause shoulder injury?"

2. This study only incorporated the involved dominant shoulder of each subject. An interesting area of investigation is in what affects may exist in the uninvolved non-dominant shoulder of the unstable shoulder.

3. Due to the potential complications specific to laxity and potential hormonal influences, females were not investigated in this project. Identifying if these same changes are evident in the female is of interest.

4. Although increases in reflex latency were noted in the unstable shoulder. We did not investigate to what amplitude must a reflex response be in order to overcome the stresses of functional shoulder activity. It may be the aim of future research to determine the appropriate amplitude of responses to thwart the effects of functional shoulder instability.

5. The ultimate course for this research to follow is that of rehabilitative protocols. Now that a specific criterion for the unstable shoulder exists (reflex stabilization, latency) the question is: "How do we decrease the effects of injury and decrease the latencies noted in the involved populations?" Identifying a protocol that is effective in rehabilitating this portion of the paradigm may prove to be significant in the prevention of functional instability.

APPENDIX A
ELECTRODE PLACEMENT SPECIFICATIONS

Below is a listing of specifications for both fine wire and surface electrode placement:

Subscapularis: For the approach to the subscapularis, the medial and lateral ends of the spine of the scapula will be marked and the midpoint will be identified. From a point 3cm below the medial border of the spine, the needle will be inserted and directed toward the midpoint of the spine.

Teres Major: The needle will be inserted along the lateral lower border of the scapula (lateral and rostral to the inferior angle.)

Supraspinatus: The needle will be inserted into the supraspinous fossa just above the spine of the scapula.

Infraspinatus: The surface electrode will be placed directly over the infraspinous fossa 2-4 cm below the medial one-third of the spine of the scapula.

APPENDIX B
INFORMED CONSENT FOR SUBJECT PARTICIPATION

IRB# 144-2002

Informed Consent to Participate in Research

You are being asked to take part in a research study. This form provides you with information about the study. The Principal Investigator (the person in charge of this research) or a representative of the Principal Investigator will also describe this study to you and answer all of your questions. Before you decide whether or not to take part, read the information below and ask questions about anything you do not understand. Your participation is entirely voluntary.

1. Name of Participant ("Study Subject")

_____

2. Title of Research Study

Muscle latencies in response to an external rotation perturbation of the glenohumeral joint

3. Principal Investigator and Telephone Number(s)

Brian M. Hatzel, MS, ATC/L, LMT	(W) 375-4683 ext. 5131; (H) 331-0988
MaryBeth Horodyski, EdD, ATC/L	(W) 392-0584 ext. 1261
Thomas Kaminski, PhD, ATC/L	(W) 392-0584 ext. 1297
Keith Meister, MD	(W) 265-0680 ext. 43024
Denis Brunt, EdD, PT	(W) 265-0085
Michael Powers, PhD, ATC/L, CSCS	(W) 392-0585 ext. 1332

4. Source of Funding or Other Material Support

None

5. What is the purpose of this research study?

The purpose of this study is to determine what differences may exist in the muscles of people with shoulder problems in comparison with people without shoulder problems. Doctors feel that making your shoulder stronger will help avoid shoulder injury in the future, in addition they feel that having fast reflexes in your shoulder will also help you stay healthy. Specifically, our intent is to see if a difference exists in the muscles of people with shoulder problems, in order to help target exercises that will help keep your shoulder in the joint properly.

6. What will be done if you take part in this research study?

If you agree to participate in this study, you will be asked to report once to the Physical Therapy Education Building in Gainesville for approximately two hours. Before the actual testing session, your height, weight, age, and which shoulder you use the most will be recorded. You will also be looked at to make sure you do not have any shoulder or arm injuries. This exam will consist of a visual check for swelling and bruising. You will also be asked if you have any soreness around your shoulder and upper arm. If you are currently experiencing any pain or injury to your shoulder, you will not be able to participate in the study. If you have ever hurt your shoulder before and it has come out of joint or if it pops out sometimes and goes back in, but are in no pain or discomfort then you will be included in the instability group. If you have no pain and are not currently injured, we will measure to see how much motion you have in your shoulders.

a. To start, you will be placed in a stable chair and your arm and shoulder will be placed in a device. This will help you get used to the test. Once you are in the device, your arm and shoulder will be moved about one to two inches. Once you understand the test we will move on to the next step.

b. At this point, thin wires will be placed into four different muscles on the shoulder that you use the most. These wires will be placed into each muscle using a needle that is approximately 4 cm long. The area of skin where the needle will be placed will first be cleansed and then numbed with a spray so you can't feel the needle. The exposed ends of the wires will be attached to a small skin sensor that will be taped to your shoulder with foam cling wrap and adhesive tape. These sensors will tell us how well your muscles are working.

c. In addition, three skin sensors will be placed on top of different muscles on the shoulder you use the most. The skin under these sensors will first be cleaned with alcohol. Paste will be applied to the sensors, which will be held in place with adhesive tape. These sensors will tell us how well your muscles are working.

d. Once all of the sensors are in place we will ask you to move your shoulder around in certain directions. By doing this we can tell if the sensors are in the right place.

e. After making sure that the sensors are in the right place we will ask you to contract your muscles. This will help us see how strong you are.

f. At this point we will once again ask you to sit in the chair while the machine moves your arm. The machine will make your arm move without you knowing. You will be asked to push against the movement the machine made. When your arm is being moved the machine has stops that will limit your movement, this will make sure that you don't hurt your shoulder. We will test your shoulder three times. During the testing we will watch to see how much and how fast your muscles are working.

g. After the movements we will remove all of the sensors and wires from you.

7. What are the possible discomforts and risks?

You may feel some discomfort as the needles are inserted into your shoulder. This discomfort will be equivalent to any previous injection you may have had. A spray to numb the skin will minimize this discomfort. During the testing trials you may feel some slight discomfort from the sensors. In addition, there is a possibility of developing a slight rash as a result of the adhesive tape used to attach the sensors. This is a rare occurrence and resolves within 1 or 2 days. If your shoulder has ever come out before you may be fearful of the machine's movement hurting your shoulder. But because the movement is restricted, the chance of your shoulder being injured is very low.

The risks of placing thin wires into your skin include discomfort at the site of puncture, possible bruising and swelling around the puncture site; rarely an infection; and, uncommonly, faintness from the procedure. If you wish to discuss this discomfort and risk you may call the Principal Investigator (Brian Hatzel) at (W) 375-4683 ext. 5131 or (H) 331-0988.

8a. What are the possible benefits to you?

You will not experience any direct benefits by taking part in this research study, however, this investigation will help advance the study of shoulder problems. This investigation of shoulder rehabilitation exercises will contribute to the body of knowledge with the intention of publishing the results in a manuscript. If the results are published, your name will not be revealed.

You will also be able to speak with the principal investigator about the results from all tests performed.

8b. What are the possible benefits to others?

The information obtained from this study may help improve the treatment of shoulder instability in the future.

9. If you choose to take part in this research study, will it cost you anything?

There will be no cost to you.

10. Will you receive compensation for taking part in this research study?

You will not be compensated for participation in this study.

11. What if you are injured because of the study?

If you experience an injury that is directly caused by this study, only professional medical care that you receive at the University of Florida Health Science Center will be provided without charge. However, hospital expenses will have to be paid by you or your insurance provider. No other compensation is offered.

12. What other options or treatments are available if you do not want to be in this study?

Participation in this study is entirely voluntary. You are free to refuse to be in the study, and your refusal will not influence current or future health care you receive at this institution.

If you are a student, you have been invited to participate in this research project because you may be interested in the project. The investigators associated with this project may or may not teach in your college or be associated with courses for which you are enrolled or might be expected to register in the future. Your participation in this study is voluntary and any decision to take part or not to participate will in no way affect your grade or class standing.

If you believe that your participation in this study or your decision to withdraw from or to not participate in this study has improperly affected your grade(s), you should discuss this with the dean of your college or you may contact the IRB office.

13a. Can you withdraw from this research study?

If you wish to stop your participation in this research study for any reason, you should contact: Brian Hatzel, MS, ATC/L, LMT at (352) 375-4683 ext. 5131 or 331-0988. You are free to withdraw your consent and stop participation in this research study at any time without penalty or loss of benefits to which you are otherwise entitled. Throughout the study, the researchers will notify you of new information that may become available and that might affect your decision to remain in the study.

In addition, if you have any questions regarding your rights as a research subject, you may phone the Institutional Review Board (IRB) office at (352) 846-1494.

13b. If you withdraw, can information about you still be used and/or collected?

If you wish to withdraw from the study, no information about you will be used.

13c. Can the Principal Investigator withdraw you from this research study?

You may be withdrawn from the study without your consent for the following reasons:

1. You do not qualify to be in the study because you do not meet the study requirements. Ask the Principal Investigator if you would like more information about this.
2. The investigator decides that continuing in the study would be harmful to you.
3. Study treatments have a bad effect on you.
4. Other protocol-specific reasons, for example: If you have pain in your shoulder.

14. How will your privacy and the confidentiality of your research records be protected?

Authorized persons from the University of Florida, the hospital or clinic (if any) involved in this research, and the Institutional Review Board have the legal right to review your research records and will protect the confidentiality of those records to the extent permitted by law. If the research project is sponsored or if it is being conducted under the authority of the United States Food and Drug Administration (FDA), then the sponsor, the sponsor's agent, and the FDA also have the legal right to review your research records. Otherwise, your research records will not be released without your consent unless required by law or a court order.

If the results of this research are published or presented at scientific meetings, your identity will not be disclosed.

15. How will the researcher(s) benefit from your being in this study?

In general, presenting research results helps the career of a scientist. Therefore, the Principal Investigator may benefit if the results of this study are presented at scientific meetings or in scientific journals.

16. Signatures

As a representative of this study, I have explained to the participant the purpose, the procedures, the possible benefits, and the risks of this research study; the alternatives to being in the study; and how privacy will be protected:

_____

Signature of Person Obtaining Consent Date

You have been informed about this study's purpose, procedures, possible benefits, and risks; the alternatives to being in the study; and how your privacy will be protected. You have received a copy of this Form. You have been given the opportunity to ask questions before you sign, and you have been told that you can ask other questions at any time.

You voluntarily agree to participate in this study. By signing this form, you are not waiving any of your legal rights.

_____

Signature of Person Consenting Date

APPENDIX C
MEANS FOR MUSCLE LATENCY USED IN DATA ANALYSIS

irsub	irsup	irmaj	irinf	ersub	ersup	ermaj	erinf
58.00	50.00	59.50	44.50	46.73	56.91	38.82	41.45
45.90	64.70	38.80	42.90	63.60	53.80	48.10	65.40
63.40	60.20	79.00	36.20	55.82	59.18	59.18	45.55
54.11	59.67	61.22	42.56	55.30	44.10	39.20	48.80
54.31	64.85	46.77	36.54	54.64	51.55	54.09	60.82
52.00	73.57	75.86	44.43	120.38	93.25	84.63	46.13
102.67	57.78	78.56	43.00	172.57	106.57	90.71	80.71
77.44	77.78	88.56	64.00	117.00	126.33	96.33	67.33
70.18	60.45	93.55	47.45	96.27	117.45	106.18	44.82
82.75	49.75	65.88	43.38	117.25	86.50	107.00	65.63

APPENDIX D
ANOVA TABLES

Tests of Within-Subjects Effects

Source		Type III SS	df	Mean Square	F	Sig.
MOVT	Sphericity Assumed	4117.146	1	4117.146	32.938	.000
	Greenhouse-Geisser	4117.146	1.000	4117.146	32.938	.000
	Huynh-Feldt	4117.146	1.000	4117.146	32.938	.000
	Lower-bound	4117.146	1.000	4117.146	32.938	.000
MOVT * GROUP	Sphericity Assumed	4713.845	1	4713.845	37.712	.000
	Greenhouse-Geisser	4713.845	1.000	4713.845	37.712	.000
	Huynh-Feldt	4713.845	1.000	4713.845	37.712	.000
	Lower-bound	4713.845	1.000	4713.845	37.712	.000
Error(MOVT)	Sphericity Assumed	999.960	8	124.995		
	Greenhouse-Geisser	999.960	8.000	124.995		
	Huynh-Feldt	999.960	8.000	124.995		
	Lower-bound	999.960	8.000	124.995		
MUSCLE	Sphericity Assumed	8336.321	3	2778.774	13.256	.000
	Greenhouse-Geisser	8336.321	1.785	4670.154	13.256	.001
	Huynh-Feldt	8336.321	2.550	3268.731	13.256	.000
	Lower-bound	8336.321	1.000	8336.321	13.256	.007
MUSCLE * GROUP	Sphericity Assumed	3805.999	3	1268.666	6.052	.003
	Greenhouse-Geisser	3805.999	1.785	2132.188	6.052	.014
	Huynh-Feldt	3805.999	2.550	1492.359	6.052	.006
	Lower-bound	3805.999	1.000	3805.999	6.052	.039
Error(MUSCLE)	Sphericity Assumed	5031.012	24	209.625		
	Greenhouse-Geisser	5031.012	14.280	352.308		
	Huynh-Feldt	5031.012	20.403	246.587		
	Lower-bound	5031.012	8.000	628.876		
MOVT * MUSCLE	Sphericity Assumed	1105.657	3	368.552	3.676	.026
	Greenhouse-Geisser	1105.657	2.278	485.412	3.676	.041
	Huynh-Feldt	1105.657	3.000	368.552	3.676	.026
	Lower-bound	1105.657	1.000	1105.657	3.676	.091
MOVT * MUSCLE * GROUP	Sphericity Assumed	1936.592	3	645.531	6.439	.002
	Greenhouse-Geisser	1936.592	2.278	850.214	6.439	.006
	Huynh-Feldt	1936.592	3.000	645.531	6.439	.002
	Lower-bound	1936.592	1.000	1936.592	6.439	.035
Error(MOVT*MUSCLE)	Sphericity Assumed	2406.126	24	100.255		
	Greenhouse-Geisser	2406.126	18.222	132.044		
	Huynh-Feldt	2406.126	24.000	100.255		
	Lower-bound	2406.126	8.000	300.766		

Tests of Between-Subjects Effects

Source	Type III SS	df	Mean Square	F	Sig.
Intercept	364263.667	1	364263.667	1314.381	.000
GROUP	17579.808	1	17579.808	63.434	.000
Error	2217.096	8	277.137		

APPENDIX E
TUKEY HSD POST-HOC ANALYSES: CALCULATION OF CRITICAL VALUES

q = value used to determine critical value of the Studentized Range Distribution, based on the number of means and degrees of freedom corresponding to the denominator of the significant F

Critical Value $= q \sqrt{(MS_{error} \div N)}$
N = total 3 of scores that make up the mean
X = # of means

Movement x group

N = 20; X = 4; MS_{error} = 124.99; df_{error} = 8; Table = 4.53

4.53 √ (124.99 ÷ 20) = **11.32** critical value

Muscle x group

N = 10; X = 8; MS_{error} = 209.625; df_{error} = 24; Table = 4.68

4.68 √ (209.625 ÷ 10) = **21.43** critical value

Muscle x movement

N = 10; X = 8; MS_{error} = 100.255; df_{error} = 24; Table = 4.68

4.68 √ (100.255 ÷ 10) = **14.82** critical value

Movement x muscle x group

N = 5; X = 16; MS_{error} = 100.255; df_{error} = 24; Table = 5.10

5.10 √ (100.255 ÷ 5) = **22.84** critical value

APPENDIX F
DEPENDENT T - TEST INFORMATION

Paired Samples Correlations				
		N	Correlation	Sig.
Pair 1	HT & HT2	5	0	1
Pair 2	AGE & AGE2	5	-0.626	0.259
Pair 3	WT & WT2	5	-0.456	0.44

Paired Samples Test									
		Paired Differences							
		Mean	Std. Dev	Std. Error Mean	95% Confidence Interval of the Difference		t	df	Sig. (2-tailed)
					Lower	Upper			
Pair 1	HT - HT2	0	2	0.894	-2.483	2.483	0	4	1
Pair 2	AGE - AGE2	-0.4	4.669	2.088	-6.197	5.397	-0.192	4	0.857
Pair 3	WT - WT2	-3	48.81	21.83	-63.607	57.607	-0.137	4	0.897

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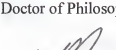
BIOGRAPHICAL SKETCH

Born in Morristown, NJ, and raised in Central Florida, Brian Michael Hatzel received his Bachelor of Science degree in Exercise and Sport Sciences with a specialization in athletic training from The University of Florida in 1997, graduating with honors. Following his aspirations of attaining a career in athletic training he pursued a professional position within the Baltimore Orioles professional baseball organization. Realizing the importance of furthering his education, Brian pursued a graduate assistantship at Ball State University, Muncie, IN, where he received a Master of Science degree in biomechanics. After two years of graduate school and working simultaneously with the Baltimore Orioles, he decided to pursue a career in academia. In 1999, Brian began his pursuit of a PhD in health and human performance with emphasis in the newly developed athletic training/sports medicine program at the University of Florida. Brian is currently serving as Assistant Professor and Clinical Coordinator for the Athletic Training Education Program in the Department of Movement Science at Grand Valley State University.

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.



MaryBeth Horodyski, Chair
Associate Professor of Exercise and Sport Sciences

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Michael Powers
Assistant Professor of Exercise and Sport Sciences

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

Keith Meister
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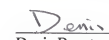

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This dissertation was submitted to the Graduate Faculty of the College of Health and Human Performance and to the Graduate School and was accepted as partial fulfillment of the requirements for the degree of Doctor of Philosophy.

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